# SDMS US EPA Region V

*Imagery Insert Form*

**Document ID:** 177713

**Some images in this document may be illegible or unavailable in SDMS. Please see reason(s) indicated below:**

[X] Illegible due to bad source documents. Image(s) in SDMS is equivalent to hard copy.

**Specify Type of Document(s) / Comments:**

SOME PAGES COPIED OFF BALANCE; SOME TEXT CUT OFF AS A RESULT

[ ] Includes _____ COLOR or _____ RESOLUTION variations.
Unless otherwise noted, these pages are available in monochrome. The source document page(s) is more legible than the images. The original document is available for viewing at the Superfund Records Center.

**Specify Type of Document(s) / Comments:**

[ ] Confidential Business Information (CBI).
This document contains highly sensitive information. Due to confidentiality, materials with such information are not available in SDMS. You may contact the EPA Superfund Records Manager if you wish to view this document.

**Specify Type of Document(s) / Comments:**

[ ] Unscannable Material:
Oversized ___ or ___ Format.
Due to certain scanning equipment capability limitations, the document page(s) is not available in SDMS. The original document is available for viewing at the Superfund Records center.

**Specify Type of Document(s) / Comments:**

[ ] Document is available at the EPA Region 5 Records Center.

**Specify Type of Document(s) / Comments:**

Rev. 07/10/02

# Assessing the Contribution from Lead in Mining Wastes to Blood Lead

By

Martha J. Steele*
Barbara D. Beck
Brian L. Murphy

*Gradient Corporation
44 Brattle Street
Cambridge, MA 02138

Harlee S. Strauss
Strauss Associates
Natick, MA

Short Title Form: Lead in Mining Wastes

*Author to whom correspondence should be sent

## Abstract

Lead has been recognized for years as an environmental pollutant of concern for young children. Nonetheless, many children in the U.S. still experience high body burdens of lead. Reducing exposure to lead must include an assessment of all potential sources of lead and a definition of routes of exposure.

In this paper, the relationships between soil lead and blood lead concentrations in residents in communities with high soil lead concentrations resulting from past mining and ore processing (milling) activities are compared to those derived from studies in urban communities or communities with operating smelters. The impact of mine waste-derived lead in soil (usually in the form of lead sulfide) on blood lead is less than for lead in soil derived from smelter, vehicle, or paint sources. Possible reasons for a reduced impact of lead sulfide on blood lead in children in mining communities include the following: lead from mining sources contributes less to lead in the immediate environment of children than lead from other sources; mine wastes typically have larger particle sizes which decrease the bioavailability of lead in the gastrointestinal tract; and lead sulfide is absorbed less in the gastrointestinal tract compared to other lead species.

A reduced impact of mine waste-derived lead on blood lead may be important from a regulatory point of view. Expensive clean-up actions for lead contaminated soils in mining

communities based on acceptable soil lead concentrations derived from smelter or urba

communities may be questionable in terms of reducing blood lead in children.

# Introduction

Lead poisoning in young children is one of the most prevalent and preventable childhood public health problems in the U.S. today (USDHHS, 1985). Lead is a ubiquitous contaminant in the environment and can enter the body from many sources including paint, air, water, soils, and foods. The relative importance of the different sources of lead exposure is a function of several variables, such as the concentration of lead in the source, the physical and chemical form of the lead, and the particular characteristics of the population of concern. Young children are more susceptible to effects from lead exposure due to physiological reasons, such as their greater ability to absorb lead from the gut, and behavioral reasons, such as greater hand-to-mouth activities (U.S. EPA, 1986a & 1986b).

In recent years, lead concentrations in some environmental media, such as air, have declined. Soils contaminated with lead, however, remain a persistent problem because of the long half-life of lead in soils. Thus, lead-contaminated soils and housedust have emerged as important contributors to blood lead concentrations. This paper reviews the contribution of lead from mine wastes in soils or housedust to blood lead levels of children living near the wastes.

Evaluating exposure to lead in mine wastes is important because regulatory actions in towns with mine wastes are often based on the predicted health risks from exposure to

soils contaminated with mining derived metals. In the case of lead, most information on the relationship between blood lead and lead in soils is derived from studies conducted in urban communities or communities with operating smelters. Based largely on these types of studies, for example, the U.S. Centers for Disease Control (CDC) has suggested that when soil lead exceeds 500-1,000 ppm, children's blood leads may increase above background levels (U.S. DHHS, 1985). The current literature suggests, however, that children living in mining towns without a recent history of smelting activities, in some cases with soil lead concentrations in excess of 10,000 ppm, do not suffer from elevated blood lead concentrations (i.e., greater than 25 $\mu$g lead/dl blood) (Heyworth et al., 1981). This observation raises the question of whether lead in mine wastes may differ in terms of public health risks from lead from other sources, such as smelters, paint, or vehicles. If so, the effectiveness of extensive and expensive removal actions of soils with high concentrations of mining-derived lead in reducing blood lead concentrations is questionable.

In this paper, epidemiological studies that have investigated the relationship between soil/dust lead and blood lead concentrations in urban towns and towns with operating smelters will be summarized to provide comparisons for a more in-depth review of the same relationship from studies in towns with mine wastes (hereinafter referred to as "mining" towns). Selected occupational health studies involving exposure to lead sulfide (galena), the primary form of mined lead, are then reviewed. These studies can be instructive in evaluating the bioavailability of lead sulfide. Finally, possible explanations

of why exposure to lead in mine wastes results in less than expected blood lead concentrations given the magnitude of soil lead concentrations will be explored. Such explanations include the following:

a. differences in the extent of migration of lead contaminated particles from the location of the mine wastes to the property of the home or inside the home, and eventually onto the hands of children residing in the home;

b. influence of particle size of mine wastes on the solubility of the lead species; and

c. the significance of the solubilities of different forms of lead in the human system, thereby affecting the bioavailability of lead in the body.

## I. Blood Lead Studies in Urban and Smelter Areas

Over the past 20 years, many investigators have studied the correlations between blood lead and environmental lead concentrations, including soil and housedust lead, especially in urban communities or communities with operating smelters. In this paper, findings from these studies will only be summarized; more detailed reviews are available elsewhere (Elwood, 1986; Duggan and Inskip, 1985; U.S. EPA, 1986a). Of particular interest are

the "slope" values derived from these studies. The slope value is the relationship of the expected increase in blood lead levels to a certain increase in soil (or housedust) lead concentration. Usually, the units are μg/dl blood lead per 1000 ppm soil lead. As stated earlier, summarizing smelter/urban studies is necessary in order to provide a comparison point for mining studies reviewed below.

A. Smelter Studies: Studies investigating the relationship between soil lead and blood lead in towns with operating smelters show a range of slopes of about 1 to 7.6 with an arithmetic mean of 4.2 and a median of 4.6, that is, an increase of 1 to 7.6 μg/dl in blood lead per 1000 ppm soil lead might be expected (Panhandle District et al., 1986; Yankel et al., 1977; Angle and McIntire, 1979; Neri et al., 1978; Walter, 1980; and Roberts et al., 1974). Table 1 shows the slopes derived for the above studies. Most of the slopes shown in Table 1 were calculated by EPA staff (U.S. EPA, 1986b). Because individuals are exposed to lead from multiple sources, simply calculating the relationship between one source and blood lead may be misleading. For example, if two sources covary, the predicted impact of one source on blood lead would be greater than the impact that actually occurs. A number of statistical models have been developed to estimate the relationship while considering other sources of exposure. EPA staff in most cases used a linear (or goodness-of-fit) model (assuming a log normal distribution for blood leads, which is consistent with many blood lead studies) to evaluate how lead concentrations in various

environmental media are separately accounted for in terms of their specific contribution to blood lead. A basic linear model is:

$$\ln (PbB) = \ln (b_0 + b_1E_1 + .... b_sE_s)$$

where: PbB = blood lead

$b_s$ = regression coefficient for source S

$E_s$ = environmental exposure from source S

$b_0 = ?$

MS

For a more detailed discussion, see U.S. EPA, 1986b.

In one study shown in Table 1 (Roberts et al., 1974), independent reviewers calculated slopes, noting that the slopes are corrected for the increase in blood lead due to air lead (Duggan and Inskip, 1985). The estimated slope of a second study (Panhandle District et al., 1986) reflects the change in blood lead vs. the change in soil lead in children living near vs. far from a smelter which was closed 2 years prior to the study, and does not take into account other factors. Thus, the slope value of 3.0 from this study may be an overestimate. All the slope values shown in Table 1 are based on geometric mean blood lead and soil lead values.*

*Population studies of blood lead concentrations show that these concentrations have a skewed distribution. Thus, it is generally more appropriate to describe the overall population blood lead concentrations in terms of geometric means, which are better to use with skewed data. It would be inappropriate to compare a geometric mean value with an

B. Urban Studies: A number of studies have been conducted in urban areas wi
smelter activities (Galke, 1975; Stark et al., 1982; Bornschein et al., 1986; Bornsche
al., 1988; Reeves et al., 1982; Rabinowitz et al., 1985; and Minnesota, 1987). In ur
areas, the main sources of blood lead are house paint and auto emissions. As in stuc
in communities with operating smelters, the range in blood lead:soil lead slopes is qu
wide. Table 2 details the estimated slopes from these studies. (All but Rabinowitz et
are based on geometric mean blood lead and soil lead values.) Two slopes were calculate
by EPA staff (U.S. EPA, 1986a) using the approach described above. One slope wa
calculated taking into account air lead exposures (Reeves et al., 1982). In the Cincinnati
studies, the slopes were calculated by the authors. Detailed data from only the 1986 study
were available. The authors developed a logarithmic "structural equations" analysis. The
equations developed are as follows:

$$\text{Ln(PbB)} = 1.276 + .152\ \text{Ln(PbH)} + .182\ \text{Ln(PbD)}$$

$$\text{Ln(PbH)} = -0.966 + .444\ \text{Ln(PbD)}$$

$$\text{Ln(PbD)} = 4.691 + .325\ \text{Ln(XRFHAZ)} + .268\ \text{Ln(PbSS)}.$$

MS Sometime pls explain diff bet a structural equation + a linear equation to me Thanks PB

arithmetic mean value. Thus, all comparisons in this paper between blood lead concentrations seen in one study vs. another study are comparisons based on geometric means.

where: PbB = blood lead

PbH = hand lead

PbD = interior dust lead

XRFHAZ = interior paint lead

PbSS = surface soil scrapings

Their 1986 data indicated that PbB increases from 10.4 μg/dl to 16.6 μg/dl with PbSS increasing from 0 to 1000 ppm. This translates to a slope of 6.2. However, when soil concentrations increased from 1000 to 2000 ppm, the PbB increased only 0.76 μg/dl, or a slope of 0.76 (Bornschein et al., 1986).

The remaining two slopes in Table 2 of 8.1 and 2.7 simply reflect the change in blood lead vs. the change in soil lead in each of the studies. They do not, therefore, control for other possible sources of lead in the environment, some of which (e.g. air) may co-vary with soil lead. Thus, these values could be overestimates. Of the six studies based on geometric mean values, the arithmetic mean slope is 3.2, the median is 2.5, and the range is 1.2 to 6.2.

The variability from household dust:blood lead concentrations is comparable to that of the soil:blood lead relationship. As with soil lead, a major problem with studies of lead in dust in relation to body lead burdens is the absence of any current standard method for

collecting dust (or soil) samples (ATSDR, 1988). For example, methods for dust collection include vacuum samples, wipe samples, and greased plate samples. Lead concentrations in housedust vary as a function of their primary contributors (e.g., paint, gasoline, stationary source emissions). For example, some authors have noted a strong correlation between type of housing (a surrogate for lead paint) and blood lead levels (Bornschein et al., 1986; Stark et al., 1982; Elwood, 1986). Reports addressing the quantitative relationship between dust lead and blood lead show a range from well below 1 to 7 for slope values (Elwood, 1986; U.S. EPA, 1986b). Recent data by Bornschein and coworkers show that dust lead transmitted via children's hands to their mouths accounts for a significant fraction of blood lead increases. Soil lead, in turn, is a significant contributor to housedust lead (Bornschein et al., 1986).

C. Summary: For studies in both urban communities and communities with operating smelters, the range of slopes relating blood lead to soil lead is similar, that is, about 1 to 8. After review of all studies at the time, EPA determined that a reasonable estimate for the slope value was 2.0 (U.S. EPA, 1986a). The basis for this conclusion was the calculation of the median slope value from the studies EPA reviewed (U.S. EPA, 1986a).

## II. Blood Lead Studies in Mining Areas

Compared to studies conducted in urban areas and areas with operating smelters, relatively few studies have been published investigating blood lead concentrations in old mining areas contaminated with mine wastes and without a recent history or any history of smelting activities. Such areas are not uncommon in some parts of the U.S., such as southeastern Missouri (the "Missouri Lead Belt") and in parts of several western states. Some sites with mine wastes are currently being investigated, and full results are not yet available.

A. Bornschein et al., 1988: This study took place in Telluride, Colorado, which has tailings piles on the edge of town. A total of 231 individuals were tested for blood lead in 1987. Of these, 96 were children 6 years or younger. The geometric average blood lead concentration in the children was 6.1 μg/dl. Samples of soil scrapings, core soils (1" deep), household dust, interior and exterior paint, and hand wipes were taken. Soil scrapings averaged 178 ppm lead (geometric mean) with a maximum of 1895 ppm. Floor dust averaged 281 ppm, and ranged from 86 to 3165 ppm, while window sill dust averaged 567 ppm (range 42 to 147,267 ppm).

A significant correlation was seen with housedust lead and blood lead, but not directly with soil lead and blood lead. As mentioned above, soil lead contributes indirectly to blood leads via contaminating housedust and then getting onto the hands of young children. The

authors concluded that for every 100 ppm lead in soil, an increase of 0.22 μg/dl lead in blood would be predicted, corresponding to a 2.2 μg/dl increase per 1000 ppm soil lead. The analysis utilized the same structural equations discussed previously. The 2.2 slope value was determined from data where soil concentrations increased from 500 to 1000 ppm. No information on PbB increases resulting from higher lead concentrations in soil, e.g. 1000 to 2000 ppm, was provided.

B. Barltrop et al., 1988: Results from a study in the villages of Shipham and North Petherton (Somerset County), England were recently reported. In the study, blood lead was measured in 178 children, divided into high and low exposure groups. The high exposure group lived in areas with soil lead concentrations greater than 1000 ppm (mean = 1850 ppm, type of mean unspecified, likely geometric). Children in the low exposure group lived in areas with soil lead concentrations less than 1000 ppm (mean = 177 ppm). For the high exposure group, the mean housedust lead concentration was 879 ppm, and for the low exposure group, the mean housedust lead concentration was 478 ppm. Both groups had virtually identical mean blood lead concentrations, 8.9 μg/dl for the high exposure group, and 8.8 μg/dl for the low exposure group. No further information on correlations between specific children and their exposures to lead in the environment was available.

C. Gallacher et al., 1984: Children from four areas of Wales with differing degrees of environmental lead concentration were studied. Two areas were near heavy traffic, one was a rural control village, and the fourth was contaminated by spoil from lead mining in the past. The investigators found a significant difference between blood lead concentrations in 1-3 year olds in the mining town vs. the control town. The mining town had a geometric mean soil lead concentration of 1167 ppm, while the control town had a geometric mean of 79 ppm (no soil measurements were made in the traffic settings). Housedust geometric mean concentrations were 350 and 177 ppm for mining and control towns, respectively. The mining town children had a mean (type not specified) of 21.8 μg/dl blood lead, while the control children had a mean of 17.0 μg/dl blood lead (a significant difference). Children in the two traffic areas had the lowest mean blood levels (14.2 and 16 μg/dl). Because the difference between soil lead concentrations in the mining and control towns is slightly greater than 1000 ppm, this would suggest an increase of about 4.5 μg/dl blood lead per 1000 ppm soil lead, assuming no other sources of blood lead covary with soil lead.

D. Heyworth et al., 1981: This study was conducted in Northampton, Australia, where galena had been mined since 1851. Tailings from the mines have been used extensively throughout the town in foundations of buildings, driveways, school playgrounds, streets, and other public areas. Surface soil from the town boundary contained 300 ppm lead, from a recreational ground, 12,000 ppm, and from a school playground, 11,000 ppm. Tailings

themselves had lead concentrations up to 150,000 ppm. Blood leads were analyzed in 181 children aged 5-14. No significant difference was found in the blood leads of children who had been living in houses reportedly built on mill tailings (mean of 10.8 μg/dl, type of mean unspecified) vs. those who had not (mean of 12.0 μg/dl). Children who lived in Northampton had mean blood leads of 12.8 μg/dl vs. children outside of town with mean blood leads of 11.2 μg/dl, a statistically significant difference. The authors suggested the blood leads found in resident children were somewhat lower than expected (based on the Barltrop et al., 1975 study). They hypothesized that either the children were exposed to soil lead concentrations considerably less than the 1% in the playground, or the lead was relatively unavailable for absorption. The study is limited by the lack of data on actual exposures to the children, on children more sensitive to lead exposures (aged 6 or less), and on the uncertainty of responses to questions on residence on mine tailings (determined by questionnaires).

E. Barltrop et al., 1975: This study was conducted in Derbyshire, England. Eighty-two children, aged 2-3 years old, had their blood analyzed for lead. Thirty-two children lived in a low soil lead area, while 48 lived in a high soil lead area. Samples from garden soil and housedust were taken from each of the homes in the study. The geometric mean soil lead concentration in the low soil lead area was 518 ppm (range 130-3000 ppm), while in the high soil lead area, the geometric mean was 4881 ppm (range 1050-28,000 ppm). The geometric mean housedust lead in the low soil lead area was 565 ppm (range 190-

2450) while in the high soil lead area, it was 1803 ppm (range 420-25,000 ppm). The children in the low soil lead area had a geometric mean blood lead of 20.9 μg/dl, while in the high soil lead area, the geometric mean blood lead was 25.0 μg/dl.

Based on this study, the U.S. EPA calculated a slope of 0.6, that is, a 0.6 μg/dl increase per 1000 ppm increase in soil lead (U.S. EPA, 1986b). Barltrop and coworkers concluded that despite the high soil lead concentrations (a high mean of 13,969 ppm), this source of exposure contributed little to children's blood lead concentrations.

F. Barltrop et al., 1974 - This study was conducted in two towns, Matlock and Buxton, in Derbyshire, England. Blood lead concentrations were measured in children aged 2-3 years. Surface soils from the home of each child were also analyzed for their lead content. The geometric mean soil lead in Matlock was 909 ppm, while for Buxton, it was 398 ppm. Blood leads were reported separately for the spring and summer seasons. For both seasons, the geometric mean blood lead concentrations were actually lower (but not significantly so) in Matlock than in Buxton. In Matlock, the geometric mean was 20.1 μg/dl in the spring and 24.7 μg/dl in the summer, while for Buxton, it was 22.8 μg/dl and 28.1 μg/dl, respectively.

G. Other Studies: Limited results of blood lead testing in towns with soils contaminated with mining-derived materials are available for Skagway, Alaska (Alaska, 1988) and Park City, Utah (ATSDR, 1988a).

Skagway, Alaska is the site of an ore terminal. Trucks loaded with ore from a nearby mine passed through a residential area in Skagway (along State Street) on their way to the ore terminal. Testing of soil samples near the terminal and along State Street revealed high lead concentrations (mean and range unknown). For example, one sample from a street gutter had a lead concentration greater than 28,000 ppm. For that location, however, five feet inside a private property line, the lead concentration was only 672 ppm. Most residential soils in Skagway had lead concentrations less than 500 ppm. Of 20 residential soil samples along State Street, 12 were greater than 500 ppm, and 5 greater than 1000 ppm.

Skagway residents did not have elevated blood lead concentrations [for children aged 0-5, mean (unspecified type) blood lead was 6.7 $\mu$g/dl; for children aged 6-18, mean blood lead was 4.5 $\mu$g/dl]. No details were available on where the subjects lived or on the soil lead concentration at their respective residences. In addition, blood lead concentrations of children living "near" vs. "far" from the ore terminal and State Street were reviewed. No significant differences between blood lead concentrations in these two groups were found (Alaska, 1988).

Two studies have been conducted in Park City, Utah, where extensive mining and milling has occurred since the late 19th century. Residential subdivisions (Prospector Area) were constructed on mine tailings in the 1970s. The first study was conducted in 1984 (Perrotta and Stafford, undated). Children, aged 2-14, from the Prospector Area and a control area 1/2 mile north of the site were chosen for blood lead determinations. The only reported soil lead concentration for either area was a maximum value of 8000 ppm in the Prospector Area. April 1984 testing results showed children in the Prospector Area (N = 39) had an arithmetic mean blood lead of 9.3 μg/dl vs. 5.3 μg/dl for the controls (N = 9). October 1984 results showed children in the Prospector Area (N = 48) had an arithmetic mean blood lead of 10.4 μg/dl vs. 9.2 μg/dl for controls (N = 19). Neither difference between the two groups of children were significant. For the more sensitive age group of 2-5 year olds, the October results showed the arithmetic mean blood lead was 11.5 μg/dl in residents vs. 9-5 μg/dl in controls (number of 2-5 year olds not reported for either group).

In 1985, city officials covered the tailings with 6 inches of topsoil. Two years later, the Agency for Toxic Substances and Disease Registry conducted a blood lead testing program for residents in the Prospector Area ("target" area) and for residents in a "comparison" area in Park City. At the same time, the EPA measured surface soil lead concentrations in both areas. The EPA report was not available for review, but results were summarized

in the ATSDR report. Despite the 1985 action of covering the tailings with 6 inches of topsoil, surface soils in the target area had maximum lead concentrations of 2250-5840 ppm [mean (unspecified type) of 3422 ppm, range of 16-5840 ppm]. Apparently, only one surface soil sample from one site in the comparison area was analyzed. The sample contained 165 ppm lead.

The results of blood lead testing in children aged 9-71 months for the target area showed a geometric mean of 7.8 μg/dl (N = 38). For the same age group in the comparison area, the geometric blood lead mean was 4.0 μg/dl (N = 13). While the differences were statistically significant, both values are well below the CDC "level of medical concern" of 25 μg/dl, and were low compared with children in other parts of the U.S. (ATSDR, 1988a). Because no soil lead concentrations are available for individual children, a slope value cannot be calculated.

H. Summary of Data from Mining Studies: Many of the mining studies are limited by a lack of environmental data for the individual children in the study, lack of information due to final reports yet to be released, and lack of subjects in the most sensitive population group (i.e., children less than 6 years old). The picture that emerges thus far, however, is that blood leads in towns with mine waste, but no recent or current history of smelting, are in general not elevated despite some very high soil lead concentrations (for example, Heyworth et al., 1981, and ATSDR, 1988a). Slopes calculated from the mining studies

range from virtually 0 (Barltrop et al., 1988) to 4.1 (Gallacher et al., 1984). Two other studies where it was possible to estimate slopes showed values of 0.6 and 2.2. The arithmetic mean of all studies is 1.7, and the median is 1.4.

## III. Comparing Urban/Smelter Studies with Mining Studies

Comparisons between studies evaluating the relationship between soil lead and blood lead can be difficult. For example, many studies investigate dissimilar population groups with respect to age, socioeconomic status, nutritional status, and sex. Site-specific variables such as soil type and form of lead may also be different. Some studies have other major sources of lead exposure, such as ambient air (in the case of operating smelters) or vehicular traffic (in the case of some urban studies). Nonetheless, a few statements can be made.

In general, studies in mining areas have found either no strong correlation between soil lead and blood lead (Heyworth, 1981), no elevated blood lead concentrations in areas with very high soil lead concentrations (Heyworth, 1981), or slopes at the low end of the range cited by EPA (Barltrop et al., 1975; Barltrop et al., 1988; Bornschein et al., 1988). While the epidemiological studies are not conclusive, taken together, they do indicate that mining wastes may be different from other sources of lead in contributing to blood leads. The general trend in the data indicates that children's blood leads tend to be lower than

expected (based on non-mining study comparisons), especially given the very high soil lead concentrations seen in some of these studies.

Figure 1 illustrates the comparison of slopes found in mining studies vs. those found in studies in urban communities or communities with operating smelters. The slopes from mining studies average 1.7 (arithmetic mean), about half the average slopes from the smelter or urban studies. If we exclude from consideration those studies where the slope values are not corrected for any other sources of lead, the average slope value from mining studies was 0.9, vs. the average of 3.3 for urban areas and 4.4 for smelter areas.

The slope relationships between lead concentrations in soil/ housedust and blood lead concentrations described in the epidemiological studies represent empirical correlations between an environmental medium containing lead and an indicator of lead body burden. As such, these estimates make no assumptions regarding exposure rate, bioavailability of lead, or the relationship between lead uptake and blood lead concentrations.

An alternative way to predict the effect of lead exposure on blood lead concentration was developed by EPA, termed the "Integrated Uptake/Biokinetic" (IU/BK) model (U.S. EPA, 1986a). The IU/BK model represents a flexible technique for incorporating information on lead concentrations in different environmental media and predicting the impact on blood lead. The model is potentially very useful in defining the most important

environmental sources of lead. EPA staff recently validated the model for several locations and found general support for the efficacy of the model (USEPA, 1988).

The model, based on a two-year old child, was derived from assumptions about basic exposure parameters, on gastrointestinal and pulmonary lead absorption, and on the relationship between lead uptake and blood lead. The IU/BK model has ~~four~~ five basic steps:

1. Quantification of lead concentrations in different media (air, soil, housedust, diet).

2. Estimation of lead intake from that source, using standard assumptions (e.g. ingestion of 80-135 mg per day of soil/ housedust, U.S. EPA, 1988). These assumptions also include weighting factors to account for time spent outdoors vs. indoors.

3. Calculation of amount of lead absorbed, using appropriate absorption factors for children specific to the lungs or gut.

4. Calculation of total lead absorption by summing absorption from all sources.

5. Estimation of blood lead from the total lead uptake value using the biokinetic model of Harley and Kneip (1985).

The amount of lead absorbed is then converted to blood lead using data derived from lead ingestion studies in infants and toddlers (Ryu et al, 1983; Harley and Kneip, 1985). The relationship between lead uptake and blood lead is relatively linear for blood lead concentrations below 30 μg lead/DL blood. It is inappropriate to use this model in situations resulting in blood lead concentrations greater than 30 μg/DL.

The IU/BK model was also applied to blood lead data and environmental data from the Doe Run Company Smelter site in Herculaneum, Missouri (Vornberg et al., 1988). This is the site of an operating smelter (?) These authors used the EPA approach, except that an ingestion rate of 60 mg soil/housedust per day was assumed (EPA assumed 80-135 mg per day). An absorption factor of 30% was used. The relationship between the predicted and observed blood concentrations for children was good with the ratio of predicted to observed falling between 0.7 and 1.1 for 17 of the 20 locations in the 1984 survey. The predictability was comparable for different soil/dust lead concentrations ranging from 70 to about 2000 ppm lead and for different air lead concentrations ranging from 0.3 to 2.8 $\mu g/m^3$.

EPA staff recently validated the IU/BK model with data from a 1983 study in E. Helena, Montana (USEPA, 1988). Children aged 1-5 years were studied. They lived in one of three areas located at progressively greater distances from a smelter. The IU/BK model closely predicted the blood lead concentrations actually observed in the children living in Areas 1 and 2. In three separate runs, each using different methods to estimate ambient

air and/or soil/dust lead concentrations, the ratio of predicted vs. observed blood lead values ranged from 0.99-1.02. ~~Unlike the analysis of Vornberg and coworkers, who assumed 60 mg soil ingestion/day~~, EPA staff assumed 80-135 mg soil ingestion/day and ~~2x~~ 30-3C in the abov validation of the IU/BK model using E. Helena data.

→ Insert (A)

To provide a comparison to smelter communities, the ~~IU/BK model was also~~ we applied the IU/BK mod to data from mining communities (data from Barltrop et al., 1988). These investigators studied a group of 178 children in the mining communities of Shipham and North Petherton, England. Children were divided into low exposure (soil lead <1000 ppm) and high exposure (soil lead >1000 ppm) groups. In this study, greater than twofold differences in soil/ housedust lead concentrations were observed for the low and high exposure groups.

Blood lead concentrations were quantified in the two groups and compared with lead concentrations in soil and housedust:

| | Blood lead μg/dl | Soil lead ppm | Housedust lead ppm |
|---|---|---|---|
| High Exposure Group | 8.9 | 1,850 | 879 |
| | (99) | (130) | (119) |
| Low Exposure Group | 8.8 | 177 | 478 |
| | (79) | (99) | (101) |

Number in parentheses = number of cases or samples

The time weighted average soil/dust concentrations were determined for the two exposure scenarios, assuming that a child spent three hours outside and nine hours awake inside each day. This calculation yielded an average of 1122 ppm lead per day in soil/dust for the high exposure group, and 403 ppm lead per day in soil/dust for the low exposure group. Assuming 60 mg soil/dust ingested per day (from Yornberg et al, 1988) and a 25% lead absorption from the gut for lead in soil/dust (from USEPA, 1988), the daily lead uptake for the high exposure group was estimated as 17 μg, and for the low exposure group, 6 μg.

For the high exposure group, the predicted impact of the soil/dust lead on blood lead is about 8 μg/dl, while for the low exposure group it is about 4 μg/dl (based on Harley and Kneip, 1985). The actual observed blood lead in the high exposure group was 8.9 μg/dl, and for the low exposure group, 8.8 μg/dl. These observed values include exposure to lead

from all sources. In the U.S., for example, dietary lead could add another 4.0 μg lead/dL blood to the above estimates (U.S. EPA, 1988).

These calculations demonstrate that the IU/BK model overpredicted the impact of mining derived lead on blood lead concentrations in children. Furthermore, the model predicted that children in the high exposure group should have about 4 μg lead/dL blood more than children in the low exposure group. In fact, no differences were observed between the two groups.

The IU/BK model was also applied to earlier data by Barltrop and coworkers (1975), described earlier. Using ~~similar~~ the same methods for estimating lead uptake as above, 38.5 μg/day is the estimated lead uptake for the high exposure group, and 8.3 μg/day for the low exposure group. Resulting blood lead levels for these lead uptakes are approximately 16 μg/dl and 4.5 μg/dl, respectively. The observed blood lead in the high exposure group was 25 μg/dl, and 20.9 μg/dl for the low exposure group. If we assume the most important difference between two groups was soil/dust lead concentrations, the IU/BK model overpredicted the actual observed difference between the groups by a factor of 3. That is, the IU/BK model predicted a difference of 12.5 μg/dl vs. the 4.1 μg/dl observed difference.

Discrepancies between the observed blood lead concentrations in mining studies and those predicted by the model may be due to several factors. For example, the soil/dust sampling

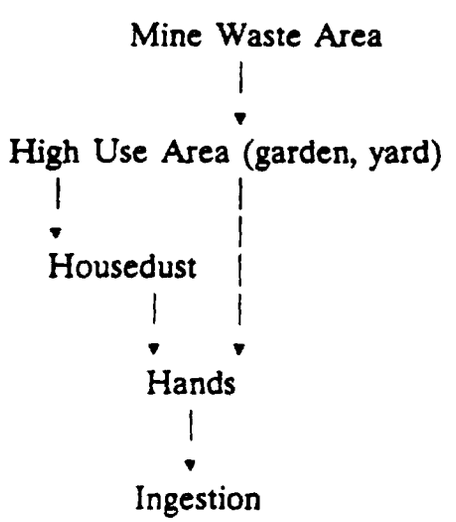
methods may not adequately reflect the soil/dust actually ingested, and the assumptions ~~on~~ for the soil ingestion rate and ~~on~~ for the percent absorption from the gut may not be accurate. Studies of soil tracer elements in feces of children median average soil ingestion rates of 50 - 110 mg/day (Clausing et al., 1987; Binder et al., 1986; Calabrese et al., 1988) with values less than 50 mg/day observed in the best calibrated study and employing the three best tracers from that study (Calabrese et al., 1988). This soil ingestion rate is lower than that used by EPA in IU/BK model (80-135 mg/day). In addition, assumptions about gastrointestinal absorption might be modified to incorporate site-specific information. Based on epidemiological studies of mining communities and on toxicological investigations of animals exposed to different lead species of varying particle sizes, a lower gastrointestinal absorption rate may be warranted for mining derived lead as discussed in later sections.

## IV. Exposure to Lead in Mine Wastes

As noted earlier, exterior soils contribute to interior housedust (Bornschein et al., 1986). Because children spend a greater proportion of time indoors than outdoors, particularly in cold weather, interior housedust can be an important direct source of lead exposure. Tracing the pathways of mine wastes from their point of origin to residential soils and then to interior housedust is of particular interest.

Two pathways may be involved when lead from mine waste sources is tracked into homes by people or animals. [Except under unusual fugitive dust conditions, air pathways into the home should be unimportant in the absence of point sources (Murphy and Yocum, 1986)]. The two pathways are: (1) infrequent traversal of mine waste areas and (2) frequent traversal of gardens and other areas near the home which may have become contaminated with mine waste materials by runoff, windblown dust, or relocation of mine wastes (see Figure 2). The tendency for particles to adhere to shoes and pets will be largely determined by moisture content of the waste and to a lesser degree by particle size and surface properties.

It is convenient to view the relationship between mine waste lead and blood lead concentrations as a series of "transfer coefficients." The concentration at each step in the chain illustrated below can be obtained from the concentration at the previous step by a multiplicative factor (less than 1):



No explicit values for a transfer coefficient between lead in mine wastes and lead in garden soil were found. It has, however, been shown that the pattern of contamination from mine wastes is distinctly different from the pattern of contamination resulting from air emissions of heavy metals from stacks (Davies and Wixson, 1985; Lagerwerff and Brower, 1975). While atmospheric contamination results in widespread uniform elevated soil concentrations exhibiting exponential decay as a function of distance from the source, the contamination from mines, ore transport, and mine wastes can be spotty. For example, in some areas where mine wastes (chat) had been used for surfacing roads, the adjacent soils had elevated lead concentrations. This effect extended only 100 meters from the road, with 50% of the contamination confined to first 25 meters (Lagerwerff and Brower, 1975).

In the Skagway investigation discussed earlier (Alaska, 1988), the limited area ~~impacted~~ affected by the transport of ore over roads was illustrated by the fact that in one soil sample from a street gutter, the lead concentration was greater than 28,000 ppm. Yet, a soil sample 5 feet within the private property line nearby showed only 672 ppm lead.

These data indicate that lead in mine wastes or mine ores may not be widely or evenly distributed from the source, especially compared to emissions from operating smelters. The particle size of mine wastes is sufficiently large such that airborne particles tend to settle out quickly and do not deposit in as broad an area as the much smaller aerosols

from stack emissions which stay airborne longer and travel farther. The particle sizes of a variety of tailings materials from different ores have been measured in the range of 10 to 1000 $\mu$m with none smaller than 1 $\mu$m (Andrews, 1975). In contrast, primary particles emitted from high energy sources such as smelters largely fall in the 1-3 $\mu$m size range, with a significant number of particles smaller than 1 $\mu$m (Perera and Ahmed, 1979).

A. Garden Soil-Housedust Transfer Coefficient: Studies conducted by Barltrop and coworkers (1975, 1988) can be used to estimate the transfer coefficients for the relationship between garden soil and housedust. Garden soil is used as a surrogate for all high use areas near the home. Table 4 shows geometric mean values for lead in housedust and garden soil in mining communities in Derbyshire (D) and Shipham/ North Petherton (SNP). The table has been constructed by aggregating the garden soil lead values in each community below 1000, between 1000 and 10,000, and above 10,000 ppm.

The data from Table 4 were plotted (using least squared regression techniques) in Figure 3. The ordinate intercept in Figure 3 of 605 ppm represents the expected background concentration of lead in housedust from sources in the house or nearer the house than the garden. The equation developed from Figure 3 is:

$$\text{Pb in housedust (ppm)} = 605 \text{ ppm} + .16 \text{ (Pb in garden soil, ppm)}$$

The value of 0.16 is the "transfer coefficient" between lead in garden soil and lead in housedust. This coefficient assumes a linear relationship between soil lead and housedust lead when concentrations of soil lead are greater than 605 ppm.

According to these results, the lead concentration in garden soil would have to be about 3900 ppm for the lead tracked in from gardens and other high use areas to contribute an amount equal to the contribution from sources, such as lead paint, in or closer to the home (e.g., 605 ppm).

Davies et al. (1985) also investigated the relationship between garden soil and housedust using paired samples from a former lead mining area in North Wales. Their data showed that 27% of the variability in housedust lead could be explained by soil lead. They also developed a regression equation on the relationship between soil and housedust lead:

$$(\log \text{lead in dust,ppm}) = 0.3\ (\log \text{lead in soil, ppm}) + 1.65\ (\log)$$

By taking the antilog of this equation and by defining the transfer coefficient as the derivative of housedust concentrations with soil concentrations [d(Pbdust)/d(Pbsoil)], the transfer coefficients for different soil lead concentrations can be calculated. A transfer coefficient of .15 was derived for a soil concentration of 620 ppm. At 1000 ppm, the transfer coefficient was .11, at 2000 ppm .07, and at 3000 ppm .05. Thus, according to

these results, the lead in soil had a limited impact on lead in housedust and the relative influence of lead in soil diminished with increasing lead concentrations. That is, the relationship between soil lead and housedust lead is non-linear, in contrast to the earlier equation developed on the basis of the Barltrop data.

Table 5 shows actual data on soil lead:housedust lead concentrations from mining communities. When soil lead is less than 500 ppm, housedust lead concentrations are greater than soil lead, indicating the greater contribution of indoor sources of lead. When soil lead is greater than 1000 ppm, however, housedust lead concentrations ranged from 18 to 48% of soil lead concentrations. Furthermore, the data in Table 5 indicate a non-linear relationship between soil lead and housedust lead, consistent with the regression equation developed by Davies and co-workers.

The relationship between lead concentrations in air and in dusts and soils in urban and smelter communities has also been examined by EPA staff (U.S. EPA, 1986). EPA's summary of the data indicates that the soil/indoor dust relationship is about 1:1, or the "transfer coefficient" between soil and housedust is generally about 1 (see Table 6). This contrasts with the data from mining studies, where housedust lead is from 18-48% of soil lead concentrations.

B. Conclusions: In conclusion, the lower than expected blood lead concentrations in mining communities may be due in part to the low contribution of lead in mine waste to lead in soils and to lead in housedust. As indicated earlier, the particle size of mine wastes is sufficiently large such that airborne particles from a mine waste source tend to settle out quickly and do not deposit in as broad an area as the much smaller aerosols from stack emissions which stay airborne longer and travel farther.

## V. Occupational Studies

Occupational studies of workers exposed to lead sulfide can be instructive in evaluating the bioavailability of lead sulfide and in providing quantitative information on exposure levels of lead particulates of defined size fractions. Thus, these studies can be compared with experimental studies of adult men exposed by inhalation to submicronic lead oxide aerosols or to lead in ambient air (mainly lead halides, lead sulfate, and lead oxide) to evaluate the bioavailability of lead sulfide as compared to other lead species.

Two studies of workers exposed to lead sulfide during milling operations are available (Belden and Garber, 1949; Roy, 1977). In the Belden and Garber study, physical exams and clinical chemistry evaluations were performed on 16 workers at 5 mills in the Missouri "Lead Belt". The investigators noted that there was nothing in the physical exam, which included indicators such as wrist drop or history of weakness, to indicate lead toxicity.

Blood cell cytology was normal with no evidence of basophilic stippling or reduced red cell counts. Finally, blood lead concentrations were between 3 and 142 $\mu$g lead, with an average (probably arithmetic) of 26 $\mu$g lead/dL of blood. (Many of these values would be considered high by today's standards.) An average value of 26 $\mu$g lead/dL blood is consistent with the lack of anemia, which has a threshold of about 50 $\mu$g lead/dL blood in adults (U.S. EPA, 1986a). Thus, the blood lead concentrations are consistent with the clinical picture.

Air lead concentrations at the four mills were extremely high, ranging from 300 to 8000 $\mu g/m^3$ air. These are at least 6 times higher than the current OSHA permissible exposure level for lead of less than 50 $\mu$g lead/$m^3$ air, which is set to protect workers against a blood lead concentration of 80 $\mu$g/dL blood at present, declining to less than 50 $\mu$g/dL blood in the fifth year of the standard (OSHA, 1987).

While the data are not sufficient to calculate an accurate slope value ($\mu$g lead/dL blood per $\mu$g lead/$m^3$) for lead sulfide, a rough estimate of the maximum slope may be calculated using the following assumptions. A geometric mean of air lead concentrations can be calculated from the highest and lowest values to yield an estimated air lead concentration of 1500 $\mu$g lead/$m^3$ air. Using an average (probably arithmetic) blood lead concentration of 26 $\mu$g/dL blood, an estimate of .017 $\mu$g blood lead/$\mu$g air lead is obtained. (Using an arithmetic mean blood level would tend to overestimate this slope value compared to the

use of a geometric mean. Information was not sufficient to calculate a geometric mean blood level.)

To convert this estimate from an 8 hour per day, 5 day per week exposure to a 24 hour continuous exposure, the slope is multiplied by 4.2 to yield a "slope" of less than .07 $\mu$g lead/dL blood per $\mu$g lead/m$^3$ air. (Note: this represents a maximum estimate, because non-occupational sources of blood lead would also have contributed to the overall blood lead concentrations.) This calculation assumes a linear relationship between exposure time and blood lead concentrations, which is reasonable for a chemical with a long pharmacological half life such as lead.

Roy (1977) studied 15 (out of a total of 24) male workers at a lead mill in the Missouri Lead Belt. Personal samplers were used to obtain total and respirable air lead concentrations. Air lead concentrations were elevated, with 85% of the measurements of total air lead exceeding the 1977 proposed OSHA standard of 100 $\mu$g/m$^3$. The arithmetic mean total air lead was 420 $\mu$g/m$^3$ and the arithmetic mean respirable air lead was 110 $\mu$g/m$^3$. The arithmetic mean blood lead concentration was 42 $\mu$g/dL.

A slope for blood lead:total air lead was calculated using the same approach as described above. This calculation yields an estimated slope of less than 0.42 $\mu$g lead/dL blood for total lead/m$^3$ air. A slope of 0.33 $\mu$g lead/dL blood per $\mu$g respirable lead/m$^3$ air was

calculated from the graphic representation of the data set for respirable air lead (Figure 4). The closeness of the slopes for total and respirable lead suggests that the bulk of the particulates are in the respirable size range and that non-occupational sources of lead are not a major contributor to blood lead in this worker population.

These slope estimates may be compared to slope values derived from experimental inhalation studies of adult men to lead oxides, halides, nitrates, and sulfates (Table 7) (U.S. EPA, 1986). These slope estimates were calculated by EPA staff and ranged from 0.8 to 2.9. For each study, an inhalation slope was calculated for each individual subject. The mean slope for the study was then calculated as the arithmetic mean of the individual slope values. The experimental inhalation slopes are from 2 to 40 fold higher than those from lead sulfide, demonstrating reduced bioavailability of inhaled lead sulfide as compared to other forms of lead commonly found in the ambient air.

Other human studies involving exposure to lead sulfide are of more limited value. For example, studies of Asians exposed inadvertently to lead sulfide through a cosmetic powder (Ali et al, 1978) and of adult volunteers ingesting lead sulfide (Rabinowitz et al, 1980) demonstrate that lead sulfide can be absorbed through the gut. Due to inadequate information on particle sizes (Ali et al 1978 and Rabinowitz et al, 1980) and on exposure levels (Ali et al, 1978), these studies are less useful than the occupational studies for evaluating the bioavailability of different forms of lead.

## VI. Intestinal Absorption of Lead in Soils

It has generally been assumed that adult humans absorb roughly 10% of the total amount of lead ingested in the diet (Barltrop and Khoo, 1975; Heard and Chamberlain, 1982). However, the level of lead absorption through the gastrointestinal tract from soils and dusts may be quite different. The major factors that affect the absorption level include: 1) the chemical form of the lead, 2) the size of the lead-containing particle, and 3) the nutrients or other compounds ingested along with the lead. The following section reviews the literature pertaining to the first two of these factors. The third factor is not addressed because data are inadequate on the relative concentrations of other soil constituents in mining vs. smelter or urban communities. Next, data on particle sizes and lead content in soils and mining wastes are compared. Finally, these differences are discussed in the context of their predicted effect on the absorption, or bioavailability, of ingested lead.

A. Effect of lead compounds and particle sizes on intestinal lead absorption: Barltrop and Meek (1975) examined the absorption of twelve different lead compounds, including solids and oily, viscous liquids, relative to lead acetate absorption. Young rats were fed a diet containing 0.075% of the indicated lead compound for 48 hours. At the end of this period, the rats were sacrificed, and the lead content of blood, femur, and kidney was determined. The absorption of metallic lead (particle size of 180-250 $\mu$m) was lower than

the absorption of lead salts (particle sizes <50 $\mu$m). Of all compounds, lead carbonate had the highest absorption which the authors suggested may reflect the greater solubility of this compound in gastric juice. Lead sulfide and lead chromate absorption was significantly less than lead acetate, while the other lead species (including lead oxides) had similar absorption as lead acetate.

In a later study, Barltrop and Meek (1979) directly examined the effect of particle size on intestinal absorption. Their results, based on the absorption of metallic lead using the experimental protocol described above, are summarized in Figure 5. A nonlinear inverse relationship between particle size and blood lead and kidney lead was observed. Thus, smaller particle sizes result in higher lead absorption. The influence of particle size is especially important with particles <100 $\mu$m in diameter.

Healy et al. (1982) measured the amount of lead dissolved <u>in vitro</u> in gastric fluid as a function of time from two lead sulfide samples A and B, with mean particle sizes of 100±20 $\mu$m and 30±20 $\mu$m, respectively. The lead in the smaller particle dissolved faster (100 minutes vs 200 minutes). After sufficient time, however, both samples achieved the same lead concentrations (see Figure 6). This is consistent with the results of Barltrop and Meek (1979), who essentially limited the time allowed for the particles of lead sulfide to dissolve to the residence time in the rat gastrointestinal tract.

Other animal studies indicate that lead sulfide may be less absorbed than other lead species. In one study, calves were fed lead in the form of phosphate, oxide, basic carbonate, and sulfide (Allcroft, 1950). The authors found lead sulfide to be "less toxic," as defined by lower kidney and blood lead levels and greater survival rates. In another study, guinea pigs were fed (in a flour vehicle) various lead compounds (Fairhall and Sayers, 1940). Lead sulfide ingestion generally resulted in less absorption (as measured by liver, kidney, and bone contents) than lead oxides and sulfates. No particle size information is presented in these two studies.

A key factor in the dissolution of lead sulfide is pH. For example, Healy et al. (1982) measured the solubility of lead sulfide (particle size approximately 90 $\mu$m) in several fluids, including water, saliva, and gastric fluid. The results are summarized in Table 8. The lead was relatively insoluble in water and saliva, but was 800 times more soluble in simulated gastric fluid.

Day et al. (1979) measured the solubility (extractability) in hydrochloric acid of lead from street dust collected in two industrial cities. The authors assumed the lead compounds were primarily oxides and halides emitted from automobiles. Under environmental conditions, these compounds can then be converted to carbonates and sulfates. The authors observed a sharp decrease in the extractability of lead with increasing pH. Less than 10% of the lead was extracted at pH 4 and above, while more than 80% was

extracted at pH 1, the nominal pH of gastric fluid. However, the significance of this value is unknown because the temperature of the extraction does not correspond to physiological conditions (37•C) and hydrochloric acid is a simplistic simulation of gastric fluids. Two other studies confirmed the high degree of lead solubilization from street dust samples at a pH of about 1 (Harrison, 1979; Duggan and Williams, 1977).

B. Particle size and composition of street dust and mine waste: Biggins and Harrison (1980) fractionated street dust from four roadside sites in England by size. They observed a general trend of increasing lead concentrations with decreasing particle size, with the highest lead concentrations being associated with the <38 $\mu$m size fraction. In view of the animal studies cited earlier, this lead particle size distribution would maximize intestinal absorption.

Que Hee et al. (1985) measured the lead content in six size fractions of housedust collected in Cincinnati, Ohio. Their results are presented in Table 9. While this study shows that lead concentration is generally independent of particle size, the bulk of the dust particles were concentrated in smaller size ranges, with 77% of the lead present in particles smaller than 149 $\mu$m. Again, the particle size distribution of lead in housedust would maximize intestinal absorption.

Mill tailings from mining sites can have a wide range of particle sizes. In a description of a copper mill in Michigan operating from the early 1900s on, final mill tailings varied from 35 to greater than 200 mesh (about 370 to 74 $\mu$m and smaller) in size (Benedict, 1955). Others have noted that particle sizes in mill tailings in the U.S. vary from 1 cm or more (produced in early base-metal jig mills) to 2 mm or less in old gold and copper stamp mills (beginning in 1860s and 1870s) to a few microns in modern milling operations (particularly, 1920s and forward). Most tailings discharged from modern mills vary from 0.2 mm to a few microns (Dean et al., 1986). It should be noted that many of the mining studies described in this paper were from communities where mining and/or milling operations did not represent the modern variety.

Particle size may also be relevant to the extent of contamination of children's hands by soil/dust lead. At a recent conference, one investigator reported that dirt and dust particles on children's hands are generally less than 100 $\mu$m in size (Chaney, 1988). The implications of this finding are twofold. First, if particles containing lead sulfide from mining sources are usually greater than 100 $\mu$m, the lead sulfide particles in mining towns are less likely to adhere to children's hands, thereby reducing actual exposure to lead. (The authors of a study discussed earlier (Bornschein et al., 1988) hypothesized that one reason elevated blood leads were not seen in young children despite high soil lead concentrations was the fact that 82% of soil lead was found on particles > 149 $\mu$m.) Secondly, the fact that soil particles on children's hands are generally less than 100 $\mu$m

in size means the lead on the particles will be more bioavailable than if children were ingesting larger sized particles.

C. Model of Particle Size and Solubility Effect on Rate of Lead Sulfide Absorption: The effect of the particle size and lead compound solubility on the rate of lead absorption can be combined in a model based on the Noyes-Whitney dissolution rate law (Healy, 1984). This model predicts that the rate of lead dissolved is directly proportional to both the solubility of the lead compound and the amount of lead on the surface (i.e., the surface area for a homogeneous particle) available for interaction with the environment. Mathematically, the equation can be written as:

$$dC/dt = k'S(C_s-C) \qquad \text{Eq. (1)}$$

where C is the concentration of lead in bulk solution, S is the surface area of the particle, $C_s$ is the solubility, and k' is a constant of proportionality. This model assumes that the particle dissolution is reversible and allows a nonzero initial concentration of lead.

For the case of lead particles dissolving in the GI tract, this model can be simplified by assuming (realistically) that lead particles cannot be reformed in the GI tract and that the lead concentration, C, is always well below the solubility limit. The assumption that lead

particles won't be reformed is reasonable because some of the solubilized lead will be absorbed into the body leading to non-equilibrium conditions.

Noting that $S \propto R^2$ and that $d/dt(CV)$ is $\propto d/dt(R^3)$, we have:

$$R = Ro - kC_st \qquad \text{Eq. (2)}$$

where R is the radius after time t in the gastrointestinal tract, Ro is the initial particle radius, $C_s$ is the solubility, and k is a constant of proportionality. The fraction of the particle dissolved during a fixed amount of time can be derived from this equation as follows:

$$f = \frac{R_o^3 - R^3}{R_o^3} = 1 - \left[1 - \frac{k C_s t}{R_o}\right]^3 \quad \text{when} \quad \frac{k C_s t}{R_o} \leq 1 \qquad \text{Eq. (3)}$$

$$= 1 \quad \text{when} \quad \frac{K C_s t}{R_o} > 1$$

For particle sizes $Ro < kC_st$, the fraction of the particle dissolved is independent of solubility. Thus, for small enough particles, or long enough time, solubilization will be complete, regardless of the solubility of the compound.

For particle sizes of $Ro >> kC_st$

$$f \approx 3\,kC_st/Ro$$

Thus, for large particles, the fraction of the particle dissolved is proportional to the solubility of the compound.

Figure 7 shows how the model for particle dissolution, as defined in Equation 3, can be fit to the experimental data from Barltrop and Meek (1979). The theoretical curve in Figure 7 was obtained assuming $kC_st$ = 16.5 to fit the data. The numerical values of 8 and 22 fit the equation to the observed increase in blood lead over the baseline of 8 μg/dl. It is assumed that there is a linear relationship between blood lead and lead dissolved in the stomach. Further experiments with lead sulfide similar to those performed by Barltrop and Meek (1979) would be very helping in developing $kC_st$ relationships for application to site-specific assessments.

D. Conclusions: Based on the data presented in this section, it appears likely that lead in mine wastes, typically in the form of lead sulfide, is less bioavailable than lead typically found in street dusts, partly due to its lower solubility. One reason may be that ingested lead sulfide is generally less absorbed in the gastrointestinal tract than other lead species. A second reason is that lead on smaller particles, particularly less than 100 μm in size, is absorbed more easily in the gastrointestinal tract. Sampling data have demonstrated that mine wastes can have particle sizes greater than 100-150 μm in size, and that most lead

is lead sulfide. For street dust, most lead is found on smaller particles (<150 $\mu$m) and is usually in the sulfate, halides, or oxide form. The experimental data presented above show that particles greater than 150 $\mu$m in size would be expected to be dissolved at a much lower rate than the 40-150 $\mu$m dust particles that contain the largest concentrations of lead.

## VII. Conclusions

This paper has reviewed studies indicating that children living in areas with high soil lead concentrations due to mine wastes appear to have lower than expected blood lead concentrations. Epidemiological studies from urban communities and/or communities with operating smelters were summarized to provide a comparison for studies in mining communities. For all studies, "slope" values were derived. The slope is the relationship of the expected increase in blood lead levels to a certain increase in soil (or housedust) lead concentration.

Slope values in urban and/or smelting communities generally ranged from approximately 1 to 8, while studies in mining communities were in the low end of that range (0 to 4). Taken together, the epidemiological data therefore indicate that lead in mine wastes may have less of an impact on the body burden of lead in children residing in the mining town as compared to other communities with lead contaminated soils. Unfortunately, very few studies have been completed on children living in mining towns with no recent smelter

activity. A caveat, therefore, in comparing the mining studies to urban and/or smelter studies is that mining studies generally have less environmental data, do not have environmental data specific to each individual child (such as soil lead in a particular child's yard), and are fewer in number.

Several possible reasons for this observation have been explored in this paper. Exposure to lead in mining communities may be reduced compared to urban/smelter communities. For example, lead in mine wastes may contribute less to lead in soils than lead from operating smelters or in urban settings. The particle size of mine wastes is sufficiently large such that airborne particles from a mine waste source tend to settle out quickly and do not deposit in as broad an area as the smaller aerosols from stack emissions which stay airborne longer and travel farther.

In urban settings or areas with operating smelters, indoor dust concentrations were similar to soil concentrations. In mining studies, however, indoor dust concentrations were less than soil concentrations, varying from about 15-45% of the soil concentration. This relationship was true only when soil concentrations were greater than about 500-1000 ppm. At lower soil concentrations, housedust concentrations were often similar to or greater than soil concentrations, probably reflecting the predominance of indoor sources to housedust lead at lower soil concentrations.

Possible reasons for lower housedust lead concentrations in mining communities include the fact that in urban communities and/or communities with operating smelters, lead from deposition of airborne lead is more concentrated on surface soils, and thus more available to be tracked into homes. In addition, airborne lead can penetrate buildings and contribute to housedust lead concentrations in this manner. Furthermore, the physical-chemical properties of lead in soil may influence its potential to be tracked into homes.

Existing data indicate that most mine wastes are primarily composed of lead sulfide. Occupational studies involving exposures to lead sulfide demonstrate that workers exposed to very high concentrations of lead sulfide did not have blood lead concentrations as high as predicted. Slope values for inhalation exposure to lead sulfide were compared with slope values from inhalation studies with other lead compounds. Slopes for lead sulfide were from 2 to 40 times lower, demonstrating reduced bioavailability of lead sulfide via inhalation exposure.

Finally, possible biological mechanisms for a reduced bioavailability of lead sulfide were reviewed. Experimental data investigating the effect of particle size and lead species solubilities were reviewed. These data indicated that small particles typical of urban street dust ($<140$ $\mu$m) dissolve more readily in the gastrointestinal tract than larger particles which may be typical of mine wastes ($>150$ $\mu$m). Experimental studies also show that lead

sulfide is less absorbed than other lead species after ingestion. This may be due to the fact that lead sulfide is less soluble than other lead species in the gastric fluid.

More research is needed on the relationship between blood lead concentrations and exposure to lead in mine wastes. For example, future epidemiological surveys should provide measurements of lead concentrations in interior housedust. This is based on evidence that exterior soils contribute to interior housedust, which in turn contributes to lead found on children's hands, ultimately resulting in inadvertent ingestion of lead by children. Recent work in Cincinnati indicates that soil surface scrapings do not correlate directly with blood leads. Instead, they correlate indirectly with blood leads via housedust. Thus, sampling of lead content of exterior soils may inadequately reflect the primary exposure of children - that of housedust. This review has presented evidence that mine wastes may contribute less to housedust than urban soils or soils contaminated with metals from smelter sources.

Other questions that need to be addressed include the following:

1. Better definition of particle size vs. lead content in housedust and soils in mining communities. Evidence exists that mine waste particle sizes are larger than those reported in soils/dusts in some urban and operating smelter community studies. If this is so, most of the lead in mining communities might be found on larger particle

sizes, which in turn makes that lead less likely to enter buildings and less bioavailable.

2. Research in animal models on the difference in uptake of lead in soils and housedust in mining communities vs. urban and operating smelter communities. This research is suggested because of the possible difference in uptake of different lead species and because different lead species may predominate in mining vs. urban and smelter communities.

In conclusion, the evidence from epidemiological studies that lead in mine wastes may be less bioavailable has both biological and physicochemical plausibility. As more studies investigating blood lead concentrations in children residing in mining towns become available, this issue should continue to be explored. Such information will help ensure that remedial activities are consistent with the public health risks posed by such sites.

## Acknowledgement

This work was performed under contract to Arco Coal Company, a division of Atlantic Richfield Company.

[1]Population studies of blood lead concentrations show that these concentrations have a skewed distribution. Thus, it is generally more appropriate to describe the overall population blood lead concentrations in terms of geometric means, which are better to use with skewed data. It would be inappropriate to compare a geometric mean value with an arithmetic mean value. Thus, all comparisons in this paper between blood lead concentrations seen in one study vs. another study are comparisons based on geometric means.

## References

Agency for Toxic Substances and Disease Registry (ATSDR). (1988). The Nature and Extent of Lead Poisoning in Children in the U.S.: A Report to Congress.

Agency for Toxic Substances and Disease Registry (ATSDR). (1988a). The Silver Creek Mine Tailings Exposure Study: Park City, Utah.

Alaska (Department of Health and Social Services, Division of Public Health). (1988). Interim Report #3: Skagway Heavy Metal Investigation.

Ali, A.R., Smales, O.R.C., and Aslam, M. (1978). Surma and lead poisoning. **Br. Med. J. 2(6142)**, 915-916.

Allcroft, R. (1950). Lead as a nutritional hazard to farm livestock. IV. Distribution of lead in the tissues of bovines after ingestion of various lead compounds. **J. Comp. Pathol. 60:190-208.**

Andrews, R.D. (1975). Tailings: Environmental Consequences and Review of Control Strategies. Paper presented at the International Conference on Heavy Metals in the Environment, Toronto, Ontario, Canada.

Angle, C., and McIntire, M.S. (1979). Environmental lead and children: the Omaha study. **J. Toxicol. Environ. Health. 5**, 855-870.

Angle, C., Marcus, A., Cheng, I., and McIntire, M.S. (1984). Omaha childhood blood lead and environmental lead: a linear total exposure model. **Environ. Res. 35**, 160-170.

Annest, J.L., Pirkle, J.L., Makuc, D., Neese, J.W., Bayse, D.D., and Kovar, M.G. (1983). Chronological trend in blood lead levels between 1976 and 1980. **New Eng. J. Med. 308**, 1373-1377.

Barltrop, D., Strehlow, C.D., Thorton, I., Webb, J.S. (1974). Significance of high soil lead concentrations for childhood lead burdens. **Env. Health Perspect., 7**, 75-82.

Barltrop, D., Thorton, I., Strehlow, C.D., and Webb, J.S. (1975). Absorption of lead from dust and soil. **Postgraduate Medical Journal, 51**, 801-804.

Barltrop, D. and Meek, F. (1975). Absorption of different lead compounds. **Postgrad Med J 51**, 805-809.

Barltrop, D. and Khoo, H. (1975). The influence of nutritional factors on lead absorption. **Postgrad Med J 51**, 795-800.

Barltrop, D. and Meek, F. (1979). Effect of particle size on lead absorption from the gut. **Arch Environ Health 34**, 280-285.

Barltrop, D., and Strehlow, C.D. (1988). The contribution from soil and housedust lead to lead burden in childhood. Paper presented at a Conference on Lead in Soil: Issues and Guidelines, March 7-9, 1988, at Chapel Hill, North Carolina.

Belden, E.A., and Garber, L.F. (1949). Health of workers exposed to galena. **J. Ind. Hygiene Toxicol. 31(6)**, 347-351.

Benedict, C. Harry (1955). Lake Superior Milling Practice: A Technical History of a Century of Copper Milling.. Houghton: The Michigan College of Mining and Technology Press.

Biggins, P. and Harrison, R. (1980). Chemical speciation of lead compounds in street dusts. **Envir Sci Tech. 14**, 336-339.

Binder, S., Sokal, D., and Maughan, D. (1986). Estimating soil ingestion: the use of tracer elements in estimating the amount of soil ingested by young children. **Arch. Env. Health.** 41(6), 341-345.

Bornschein, R.L., Clark, C.S., Grote, J., Roda, S., Peace, B., and Succop, P. (1988). Soil/lead-blood/lead relationships in an urban community and in a mining community. Paper presented at a Conference on Lead in Soil: Issues and Guidelines, March 7-9, 1988, at Chapel Hill, North Carolina.

Bornschein, R.L., Succop, P.A., Draft, K.M., Clark, C.S., Peace, B. and Hammond, P.B. (1986). Exterior surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. **In: Trace Substances in Environmental Health, II. A Symposium.** University of Missouri: Columbia.

Brunekreef, B., Verristra, S., Biersteker, K., Boleij, J.S.M. (1981). The Arnhem lead study: lead uptake by 1 to 3 year old children living in the vicinity of a secondary lead smelter in Arnhem, The Netherlands. **Environ. Res. 25**, 441-448.

Chamberlain, A.C., Heard, M.J., Little, P., Newton, D., Wells, A.C., Wiffin, R.D. (1978). Investigations into lead from motor vehicles. Harwell, United Kingdom: United Kingdom Atomic Energy Authority; report no. AERE-R9198.

Charney, F. (1982). Lead poisoning in children: the case against household dust. **In:** Lead Absorption in Children: Management, Clinical, and Environmental Aspects. Chisholm, J.J., O'Hara, D.M., eds. Baltimore: Urban and Schwarzenberg.

Clausing, P., Brunekreef, B., and van Wijnen, J.H. (1987). A method for estimating soil ingestion by children. **Intern. Arch. for Occupat. & Env. Health. 59**, 73-82.

Davies, B.E., Elwood, P.C., Gallacher, J., and Ginnever, R.C. (1985). The relationships between heavy metals in garden soils and house dusts in an old lead mining area of North Wales, Great Britain." **Environ. Pollution. 9**, 255-266.

Davies, B.E., and Wixson, B.G. (1985). Trace elements in surface soils from the mineralized area of Madison County, Missouri, USA. **J. Soil Science. 36**, 551-570.

Day, J.P., Fergusson, J.E., and Chee, T.M. (1979). Solubility and potential toxicity of lead in urban street dust. **Bull Environm. Contam. Toxicol. 23**, 497-502.

Dean K.C., Froisland, L.J. and Shirts, M.B. (1986). Utilization and stabilization of mineral wastes. U.S. Department of the Interior (Bureau of Mines). Bulletin 688.

Duggan, M.J., and Inskip, M.J. (1985). Childhood exposure to lead in surface dust and soil: a community health problem. **Public Health Rev. 13**, 1-54.

Duggan, M.J., and Williams, S. (1977). Lead-in-dust in city streets. **Sci. Total Environ. 7**, 91-97.

Elwood, P.C. (1986). The sources of lead in blood: a critical review. **Sci. Total Environ. 52**, 1-23.

Fairhall, L.T., and Sayers, R.R. (1940). The relative toxicity of lead and some of its common compounds. U.S. Public Health Service (Public Health Bulletin No. 253).

Galke, W.A., Hammer, D.I., Keil, J.E., and Lawrence, S.W. (1975). Environmental determinants of lead burdens in children. International Conference on Heavy Metals in the Environment, Toronto, Canada. **Institute for Environmental Studies 3**, 53-74.

Gallacher, J.E., Elwood, P.C., Phillips, K.M., Davies, B.E., and Jones, D.T. (1984). Relation between pica and blood lead in areas of differing lead exposure. **Arch. Dis. in Childhood. 59**, 40-44.

Griffin, T.B., Coulston, F., Wills, H., Russell, J.C., Knelson, J.H. (1975). Clinical studies on men continuously exposed to airborne particulate lead. In: Griffin, T.B., Knelson, J.H. Eds. Lead. Academic Press, New York, N.Y.

Harley, N.H., and Kneip, T.H. (1985). An integrated metabolic model for lead in humans of all ages. Final Report to the U.S. EPA, Contract No. B44899 with NYU School of Medicine, Dept. of Environmental Medicine, January 30, 1985.

Harper, M., Sullivan, K.R., and Quinn, M.J. (1987). Wind dispersal of metals from smelter waste tips and their contribution to environmental contamination. **Env. Sci. Technol. 21(5)**, 481-484.

Harrison, R.M. (1979). Toxic metals in street and household dusts. **Sci. Total Environ. 11**, 81-97.

Healy, M., Harrison, P., Aslam, M., Davis, S., and Wilson, C. (1982). Lead sulfide and traditional preparations: routes for ingestion, and solubility and reactions in gastric fluid. **J. Clin. Hosp. Pharm. 7**, 169-173.

Healy, M. (1984). Theoretical model of gastrointestinal absorption of lead. **J. Clin. and Hosp. Pharm. 9**, 257-261.

Heard, M. and Chamberlain, A. (1982). Effect of minerals and food on uptake of lead from the gastrointestinal tract in humans. **Human Toxicol 1**, 411-415.

Heyworth, F., Spickett, J., Dick, M., Margetts, B., and Armstrong, B. (1981). Tailings from a lead mine and lead levels in school children: a preliminary report. **Med. J. Australia. 2**, 232-234.

Kehoe, R.A. (1961). The metabolism of lead in man in health and disease: the normal metabolism of lead. **J.R. Inst. Public Health Hyg., 24**, 81-97.

Lagerwerff, J.V., and Brower, D.L. (1975). Source determination of heavy metal contaminants in the soil of a mine and smelter area. **Trace Substances in Environmental Health., 9**, 207-215.

Landrigan, P.J., Gelbach, S.H., Rosenblum, B.F., Shoults, J.M., Candelaria, R.M., Barthel, W.F., Liddle, J.A., Smrek, A.L., Stachling, N.W., and Sanders, J.F. (1975). Epidemic lead absorption near an ore smelter: the role of particulate lead. **N. Engl. J. Med. 292**, 123-129.

Lewis and Clark County Health Department et al. (July 1986). East Helena, Montana: Child Lead Study, Summer 1983.

Lippman, M. (1978). Respirable dust sampling. In Air Sampling Instruments, G-1 to G-23, American Conference of Governmental Industrial Hygienist, Cincinnati, Ohio.

Mahaffey, K. (1983). Biotoxicity of lead: influence of various factors. **Fed. Proc. 42**, 1730-1734.

Mahaffey, K. and Fowler, B. (1977). Effects of concurrent administration of lead, cadmium, and arsenic in the rat. **Env. Health. Perspect. 19**, 165-171.

Mielke, H.W., Anderson, J.C., Berry, K.J., et al. (1983) Lead concentrations in inner city soils as a factor in the child lead problem. **Am. J. Public Health. 73**, 1366-1369.

Miller, D. (U.S. Center for Disease Control). (September 29, 1987). Memo to Mark Bashor (ATSDR) regarding results of blood lead analyses of Walkerville children.

Minnesota Pollution Control Agency and Minnesota Department of Health. (1987). **Soil Lead Report to Minnesota State Legislature.**

Moorcroft et al. (1982). Composition of dusts and soils in an apparently uncontaminated rural village in southwest England - implications to human health. **In** Trace Substances in Environmental Health, **16** (edited by D.D. Hemphill).

Murphy, B.L. and Yocum, J.E. (1986). Migration factors for particulates entering the indoor environment. 79th Annual Meeting, Air Pollution Control Association, Minneapolis, MN. June 23-28, 1986.

Neri, L.C., Johansen, H.L., Schmitt, N., Pagan, R.T., and Hewitt, D. (1978). Blood lead levels in children in two British Columbia communities. **In** Trace Substances in Environmental Health - XII: Proceedings of University of Missouri's 12th Annual Conference on Trace Substances in Environmental Health, Columbia, MO.

Occupational Safety and Health Administration. (July 1987). Occupational Safety and Health Standards - Lead. 29 CFR Chapter XVII, Subpart Z, Section 1910.1025.

Panhandle District Health Department et al. (July 1986). Kellogg revisited - 1983: childhood blood lead and environmental status report.

Perera, F.P., and Ahmed, A.K. (1979). **Respirable Particles.** Ballinger Publishing Company, Cambridge, MA.

Perrotta, D.M., Stafford, E.W. (undated). Populations exposed to mining/milling wastes. Bureau of Epidemiology, Utah Department of Health.

Raabe, OG. (1982). Comparison of the criteria for sampling inhalable and respirable aerosols. In Inhaled Particles, Volume V, (WH Walton, ed), pgs 33-45, Pergamon Press, Ltd, Elmsford, NY.

Rabinowitz, M.B., Wetherill, G.W., Kopple, J.D. (1977). Magnitude of lead intake from respiration by normal man. **J. Lab. Clin. Med., 90**, 238-248.

Rabinowitz, M., Kopple, J., and Wetherill, G. (1980). Effects and food intake and fasting on gastrointestinal lead absorption in humans. **Am. J. Clin. Nutr. 33**, 1784-1788.

Rabinowitz, M., Leviton, A., Needleman, H., Bellinger, D., and Waternaux, C. (1985). Environmental correlates of infant blood lead levels in Boston. **Env. Res. 38**, 96-107.

Reeves, R., Kjellstrom, T., Dallow, M., and Mullins, P. (1982). Analysis of lead in blood, paint, soil, and housedust for the assessment of human lead exposure in Aukland. **NZ J. Sci. 25**, 221-227.

Roberts, T.M., Hutchinson, T.C., Paciga, J., Chattopadhyay, A., Jervis, R.E., VanLoon, J., and Parkinson, D.K. (1974). Lead contamination around secondary smelters: estimation of dispersal and accumulation by humans. **Science. 186**, 1120-1123.

Roels, H.A., Buchet, J.P., Lauwerys, R.R., et al. (1980). Exposure to lead by the oral and the pulmonary routes of children living in the vicinity of a primary lead smelter. **Environ. Res. 22**, 81-94.

Roy, B.R. (1977). Effects of particle sizes and solubilities of lead sulfide dust on mill workers. **J. Amer. Ind. Hyg. Assoc. 38**, 327-332.

Sayre, J.W., Charney, E., Vostal, J., Pless, I. (1974). House and hand dust as a potential source of childhood lead exposure. **Am. J. Dis. Child. 127**, 167-79.

Scherer, J.S. (U.S. EPA, Region VIII). (October 2, 1987.) Memo to J. Winston Porter, (U.S. EPA, Region VIII) regarding removal request and $2 million exemption for the Silver Bow Creek/Butte Site - Alice Mine/Sherman Ballfield Site, Walkerville, Silver Bow County, Montana.

Schmitt, N., Philion, J.J., Larsen, A.A., Harnadek, M., and Lynch, A.J. (1979). Surface soil as a potential source of lead exposure for young children. **CMA Journal. 121**, 1474-1478.

Shellshear, I.D., Jordan, L.D., Hogan, D.J., and Shannon, F.T. (1975). Environmental lead exposure in Christchurch children: soil lead a potential hazard. **N.Z. Med. J. 81**, 382-386.

Stark, A.D., Quah, R.F., Meigs, J.W., and DeLouise, E.R. (1982). The relationship of environmental lead to blood-lead levels in children. **Env. Res. 27**, 372-383.

Thomas, H.F., Moore, F., Welsby, E., Elwood, P.C., Firth, J.N.M. (1977). The hazard of old lead mines in Wales. **Br. J. Preventive Social Med. 31**, 265-268.

U.S. Department of Health and Human Services (Centers for Disease Control). (January 1985). Preventing Lead Poisoning in Young Children.

U.S. Environmental Protection Agency (Office of Air Quality Planning and Standards). (1986a). **Review of the National Ambient Air Quality Standards for Lead: Assessment of Scientific and Technical Information.** OAQPS Draft Staff Paper.

U.S. Environmental Protection Agency (Office of Research and Development). (1986b). **Air Quality Criteria for Lead - Volume III.** EPA-600/8-83/028cF.

U.S. Environmental Protection Agency (Office of Air Quality Planning and Standards). (1988). **Review of the National Ambient Air Quality Standards for Lead: Exposure Analysis Methodology and Validation.** OAQPS Draft Report.

Vornberg, D.L., Phillips, P.R., Lanzafame, J.M. (1988). Herculaneum lead study with a risk reduction analysis. Paper presented at a Conference on Lead in Soil: Issues and Guidelines, March 7-9, 1988, Chapel Hill, North Carolina.

Walter, S.D. (1980). Age-specific risk factors for lead absorption in children. **Arch. Environ. Health. 35(1)**, 53-58.

Yankel, A.J., von Linden, I.H., Walter, S.D. (1977). The Silver Valley lead study: the relationship between childhood blood lead levels and environmental exposure. **J. Air Poll. Cont. Assn. 27**, 763-767.

Table 1 Summary Table of Blood Lead:Soil Lead Relationship from Studies in Communities with Operating Smelters

| Author | City/Study Population | Soil Lead | Housedust Lead | Slope* |
|---|---|---|---|---|
| Angle & Mcintire, 1979 | Omaha, NB<br>Age: 1-18 yrs<br>N = 1075 | Geom x = 227 ppm<br>95%tile = 843 ppm<br>(range: 16 - 4,792 ppm) | Geom x = 337 ppm<br>95%tile = 894 ppm<br>(range: 18 - 5,571 ppm) | 6.8 [a] |
| Yankel et al. 1977 | Kellogg, ID<br>Age: 1-9 yrs<br>N = 860 | x = 7000 ppm<br>(as high as 24,000 ppm within 1 mile) | x = 11,000 ppm<br>(as high as 140,000 ppm) | 1.1 [a] |
| Panhandle District et al. 1986 | Kellogg, ID<br>Age: 1-9 yrs<br>N = 364 | Geom x = 481 ppm (far)<br>3,474 ppm (near) | Geom x = 1,138 ppm (far)<br>3,933 ppm (near) | 3.0 [b] |
| Neri et al. 1978 | Trail, British Columbia<br>Age: 1-3 yrs<br>N = 87<br>Age: 1st grade<br>N = 103 | Group x in different areas of Trail ranged from:<br>225 - 1,800 ppm | N/A | 7.6 [a] for 1-3 yrs<br>4.6 [a] for 1st graders |
| Walter 1980 | Kellogg, ID<br>Age: 1-9 yrs<br>N = 983 | Not given; presumably similar to Yankel et al, 1977 | Not given; presumably similar to Yankel et al, 1977 | 1.1 [a] average for ages 2-7 yrs |
| Roberts et al. 1974 | Toronto, Ontario<br>mixed adults/children<br>N = 80 | Group arith. x ranged from:<br>100 - 2,626 ppm | Group arith. x ranged from:<br>845 - 2,005 ppm | 5.0 [c] |

N/A not available

* defined as the increase in blood lead (ug/dl) per 1,000 ppm increase in soil lead

[a] calculated by EPA (U.S.EPA, 1986) - takes into account other sources of exposure

[b] ^PbB (ug/dl)/^PbS (ppm) - does not take into account other sources of exposure (calculated by authors)

[c] calculated by Duggan & Inskip, 1985 - corrected for increase due to inhalation of air lead

Table 2 Summary Table of Blood Lead:Soil Lead Relationship from Urban Areas Without an Operating Smelter

| Author | City/Study Population | Soil Lead | Housedust Lead | Slope* |
|---|---|---|---|---|
| Galke, 1975 | Charleston, SC<br>Age: 0-5 yrs<br>N = 194 | Geom x = 585 ppm<br>(range: 9 - 7,890 ppm) | N/A | 1.5 [a] |
| Stark et al.<br>1982 | New Haven, CT<br>Age: 0-1 yrs<br>N = 153 | Five levels of SES<br>(group mean: 233 - 1,327 ppm);<br>Seven categories of housing construction<br>(group mean: 131 - 1,300 ppm) | For levels of SES<br>(group mean: 159 - 628 ppm);<br>For housing construction categories<br>(group mean: 239 - 756 ppm) | 2.2 [a] |
| Bornschein et al.<br>1986 | Cincinnati, OH<br>Age: 1.5 yrs.<br>N = 81 | Geom x = 1,360 ppm<br>(range: 76 - 54,519 ppm) | Geom x = 900 ppm<br>(range: 82 - 13,820 ppm) | 6.2 [c]<br>from 0-1,000 ppm soil lead<br>Estimated slope: 0.76 [c]<br>from 1,000-2,000 ppm |
| Bornschein et al.<br>1988 | Cincinnati, OH<br>N/A | N/A | N/A | 1.2 [c]<br>when soil lead increased from 500-1,000 ppm |
| Reeves et al.<br>1982 | New Zealand<br>Age: 1-3 yrs<br>N = 195 | Soil lead range:<br>24 - 842 ppm | N/A | 5.5 [d] |
| Rabinowitz et al.<br>1985 | Boston, MA<br>Age: 0-2 yrs<br>N = 249 | Group mean soil ranged from 380 - 1,011 ppm | N/A | 8.1 [b] |
| Minnesota 1987 | Minneapolis-St. Paul, MN<br>Age: 0-5 yrs<br>N = 656 | (range: 0 - 30,000 ppm) | N/A | 2.7 [b] |

N/A not available

* defined as the increase in blood lead (ug/dl) per 1,000 ppm increase in soil lead

[a] calculated by EPA (U.S.EPA, 1986) - takes into account other sources of exposure

[b] ^PbB (ug/dl)/^PbS (ppm) - does not take into account other sources of exposure (calculated by authors)

[c] calculated by Bornschein et al. - takes into account other sources of exposure

[d] calculated by Duggan & Inskip, 1985 - takes into account air lead exposure

Table 3 Summary Table of Blood Lead:Soil Lead Relationship from Mining Sites

| Author | City/Study Population | Soil Lead | Housedust Lead | Slope* |
|---|---|---|---|---|
| Bornschein et al. 1988 | Telluride, CO<br>Age: <6 yrs<br>N = 94 | Geom x = 178 ppm | Geom x = 281 - 567 ppm | 2.2 [c]<br>based on increase from 500-1,000 ppm soil lead |
| Gallacher et al. 1984 | 4 areas in Wales<br>Age: 1-3 yrs<br>N = 93 | Geom x = for soil<br>road 356 ppm<br>deadend 271 ppm<br>mining 1,167 ppm<br>control 79 ppm | Geom x = for dust<br>road 202 ppm<br>deadend 177 ppm<br>mining 350 ppm<br>control 177 ppm | 4.1 [b] |
| Heyworth et al. 1981 | Northhampton, Australia<br>Age: 5-14 yrs<br>N = 81 | Soil lead at town boundary: 300 ppm<br>playground range:<br>11,000 - 12,000 ppm | N/A | No significant difference between children with homes on tailings piles vs. those who were not. PbB were significantly higher in children residing in town vs. non-residents |
| Baritrop et al. 1975 | Derbyshire, England<br>Age: 2-3 yrs<br>N = 82 | Geom x in areas with soil lead:<br><1,000 ppm 420 ppm<br>>1,000-10,000 ppm 3,390 ppm<br>>10,000 ppm 13,969 ppm | Geom x in areas with soil lead:<br><1,000 ppm 531 ppm<br>>1,000-10,000 ppm 1,564 ppm<br>>10,000 ppm 2,582 ppm | 0.6 [a] |
| Baritrop et al. 1988 | N. Petherton & Shipham, England<br>Age: 3 yrs<br>N = 178 | Geom x = soil<br>low 177 ppm<br>high 1,850 ppm | Geom x = dust<br>low 478 ppm<br>high 879 ppm | 0 [b] |

N/A not available

* defined as the increase in blood lead (ug/dl) per 1,000 ppm increase in soil lead

[a] calculated by EPA (U.S.EPA, 1986) - takes into account other sources of exposure

[b] ^PbB (ug/dl)/^PbS (ppm) - does not take into account other sources of exposure (calculated by authors)

[c] calculated by Bornschein et al. - takes into account other sources of exposure

Table 4

Geometric mean values for lead in mining communities in Derbyshire (D) and in Shipham/North Petherton (SNP)

| Concentration of lead in garden garden soil near residence | | Lead in garden soil (ppm) | Lead in housedust (ppm) |
|---|---|---|---|
| City | ppm | | |
| SNP | <1,000 | 177 | 478 |
| D | <1,000 | 420 | 531 |
| SNP | >1,000 | 1,850 | 879 |
| D | >1,000 <10,000 | 3,390 | 1,564 |
| D | >10,000 | 13,969 | 2,582 |

From:

Barltrop and Strehlow, 1988.
Barltrop et al., 1975.

Table 5

Soil Lead: Housedust Lead
in Mining Communities

(geometric $\bar{x}$, ppm)

| | Soil Pb | Housedust Pb |
|---|---|---|
| Bornschein et al., 1988 | 178 | 281 (floor)<br>567 (windowsill) |
| Gallacher et al., 1984 | 1,167 | 350 |
| Barltrop, 1975 | 420 | 531 |
| | 3,390 | 1,564 |
| | 13,969 | 2,582 |
| Barltrop, 1988 | 177 | 478 |
| | 1,850 | 879 |
| Davies, 1985 | 1,127 | 340 |

Table 6

Generalized Relationships Between Lead Concentrations in Air and in Dusts and Soil[a]

| Air Lead (μg/g) | Outdoor Soil/Dust Lead (μg/g) | | Indoor Dust Lead (μg/g) | |
|---|---|---|---|---|
| | General | Near Point Source | General | Near Point Source |
| 0 | 5-30 | 20-100 | 5-30 | 20-100 |
| 0.1 | 20-90 | 50-300 | 40-100 | 40-200 |
| 0.2 | 40-150 | 80-450 | 70-200 | 70-400 |
| 0.3 | 70-250 | 125-600 | 100-250 | 250-600 |
| 0.4 | 100-350 | 200-700 | 200-300 | 300-650 |
| 0.5 | 150-500 | 350-800 | 250-400 | 350-700 |
| 0.6 | 200-650 | 450-1000 | 300-500 | 400-750 |
| 0.7 | 250-800 | 550-1150 | 350-600 | 500-800 |
| 0.8 | 300-950 | 650-1300 | 450-700 | 600-900 |
| 1.0 | 500-1150 | 750-1450 | 525-875 | 800-1150 |
| 1.25 | 600-1250 | 850-1600 | 625-1000 | 1000-1400 |
| 1.5 | 700-1400 | 1000-1750 | 750-1150 | 1200-1700 |
| 1.75 | 775-1450 | 1075-1950 | 800-1200 | 1350-1900 |

[a]The ranges of dust and soil concentrations for each air lead level reflect differences in emission sources, distance of measurement sites from these sources (within 5 km for point sources) and characteristics of the homes (e.g., permeability, paint condition, etc.), and other variables assumed to be representative of real world conditions.

Reference: Table B-2, U.S. EPA, 1986a

Table 7

Relationship between Blood Lead Concentrations and Air Lead in Adult Males

| Form of Lead | μg Lead/dL Blood per μg Lead/$m^3$ air | Reference |
|---|---|---|
| Submicronic lead oxide | 2.4[a] | Griffin et al., 1975 |
| Ambient air (Lead halides, sulfates, and oxides) | 0.9[a] | Griffin et al., 1975 |
| Ambient air | 2.8[a] | Rabinowitz et al., 1977 |
| Submicronic car exhaust, lead oxide and lead nitrate | 2.9[a] | Chamberlain et al., 1978 |
| Submicronic lead sesquioxide | 0.8[a] | Kehoe, 1961 |
| Lead sulfide | <0.07[b] | Belden and Garber, 1949 |
| Lead sulfide, total | <0.42[b] | Roy, 1977 |
| Lead sulfide, respirable | 0.33[b] | Roy, 1977 |

[a]Mean slopes calculated from data in OAQPS Staff Paper on Lead (U.S. EPA, 1986)
[b]Calculated in this paper.

Table 8

Lead Solubility (mg/1000 $cm^3$ at 36•C) of eye cosmetic

| Sample | Medium | | |
|---|---|---|---|
| | Water | Saliva | Simulated gastric fluid |
| Lead sulfide (galena) | 1.1 | 1.6 | 892 |
| Bradford surma | 1.2 | 1.7 | 870 |
| Al kohl (Kuwait) | 1.1 | 1.7 | 805 |
| Al kohl (Saudi Arabia) | 1.1 | 1.6 | 836 |

From: Healy et al., 1982.

Table 9

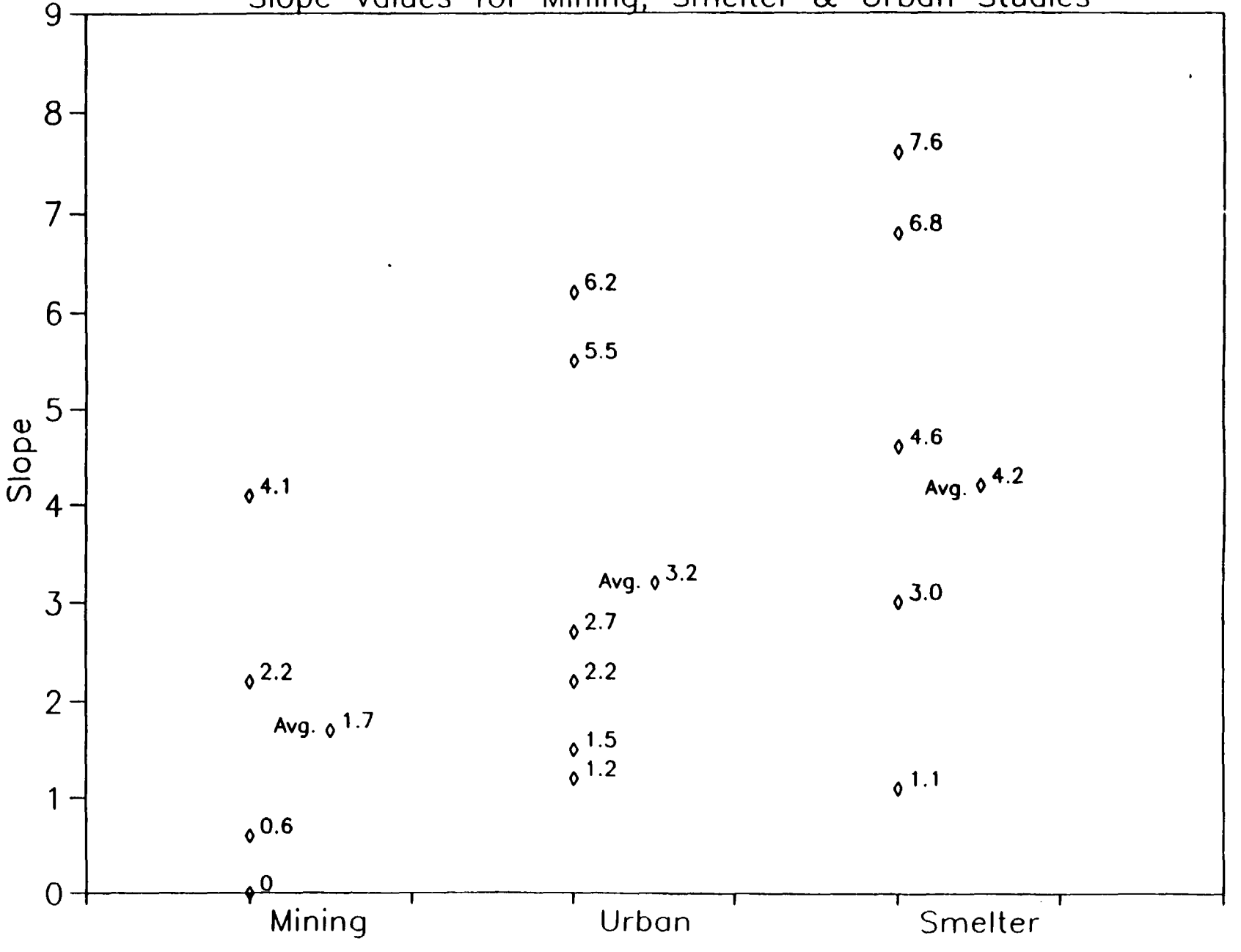
Normal Housedust Particle Size and Lead Content

| Size Range (um) | Weight % of Fractionated Dust | Lead Content μg Pb/g of Dust Fraction | % Lead in Unfractionated Dust |
|---|---|---|---|
| <44 | 18% | 1,440 | 21% |
| 44-149 | 58% | 1,180 | 56% |
| 149-177 | 4.5% | 1,330 | 4.9% |
| 177-246 | 2.7% | 1,040 | 2.3% |
| 246-392 | 6.1% | 1,110 | 5.6% |
| 392-833 | 11% | 1,090 | 9.6% |

From: Que Hee et al., 1985.

Figure 1

Slope Values for Mining, Smelter & Urban Studies[1]




1 See tables 1–3 for references

Figure 2

Sources of Lead in House Dust
In a Mining Community (No Point Sources Present)

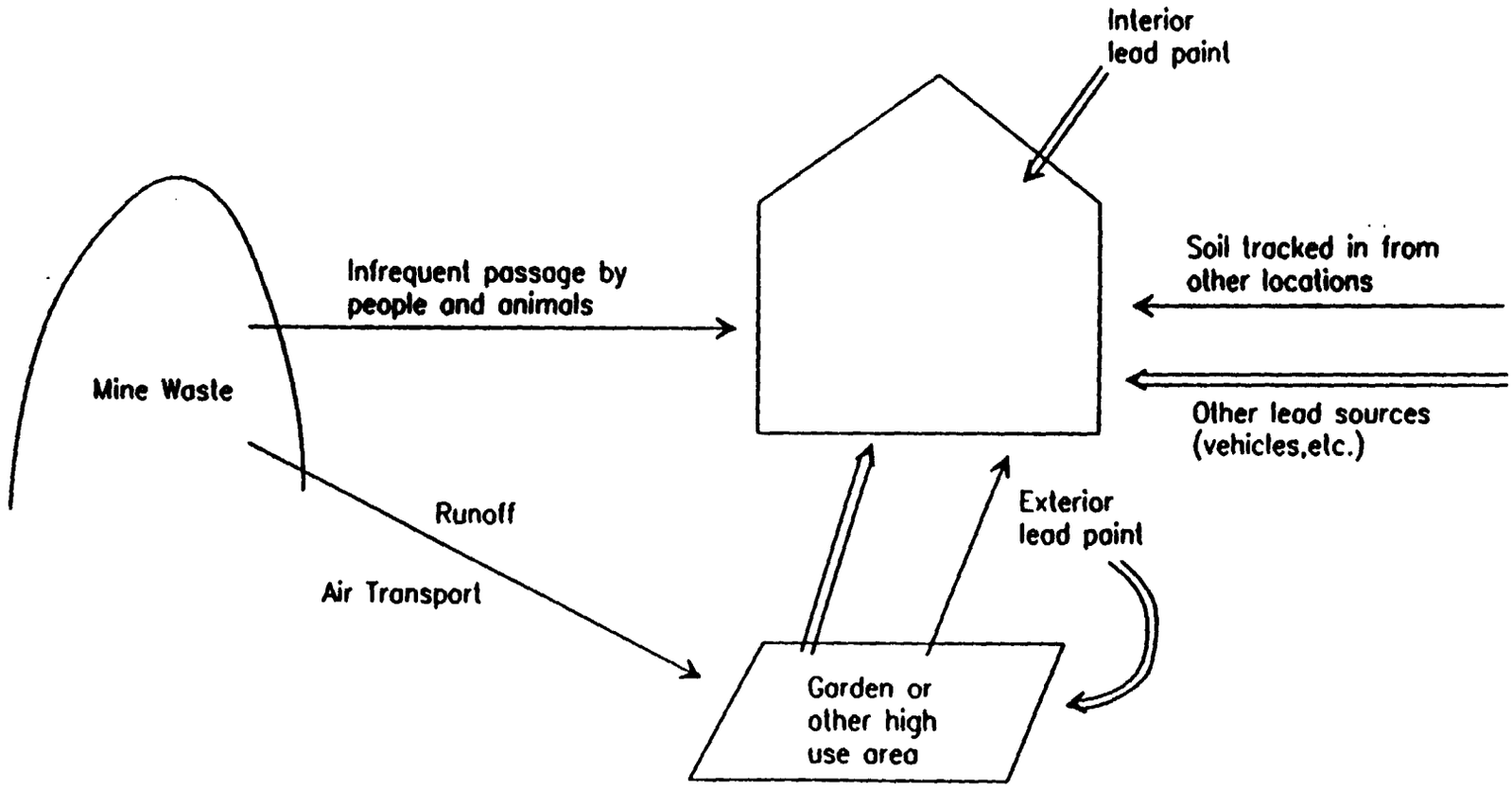
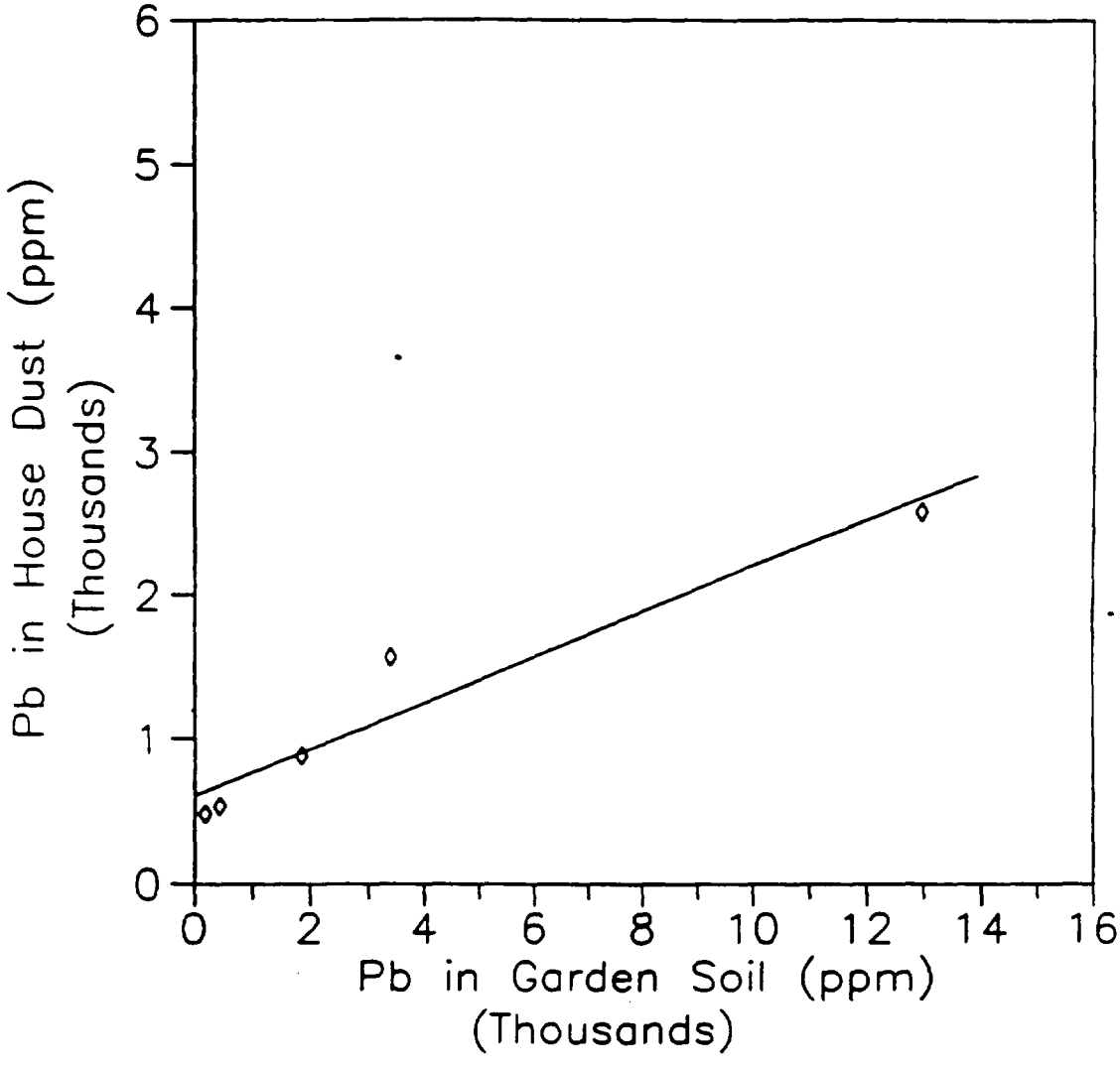


Figure 3

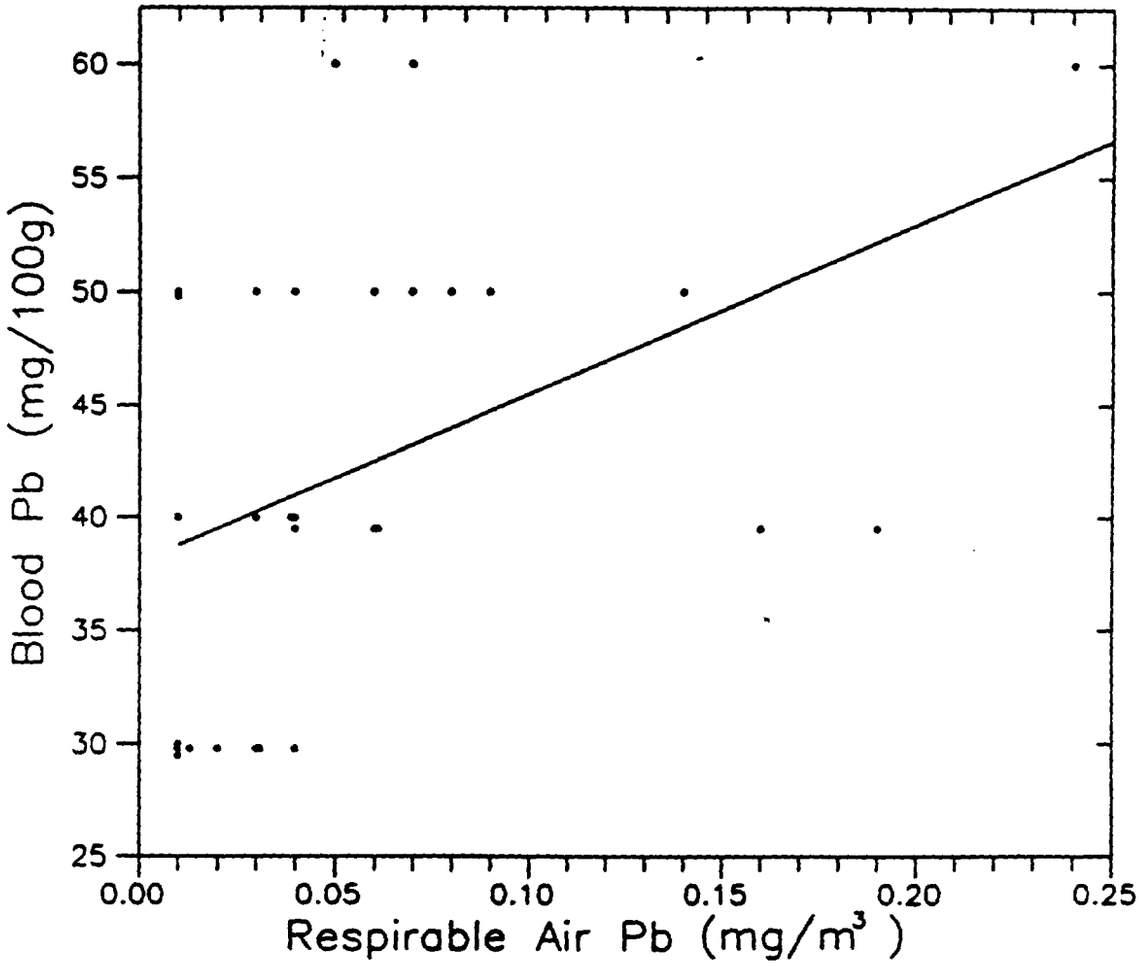
Relationship Between Garden Soil and Housedust Lead




Based on data derived from: Barltrop & Strehlow, 1988
Barltrop et al., 1975

# Figure 4

**Relationship Between Blood Lead and Respirable Air Lead**

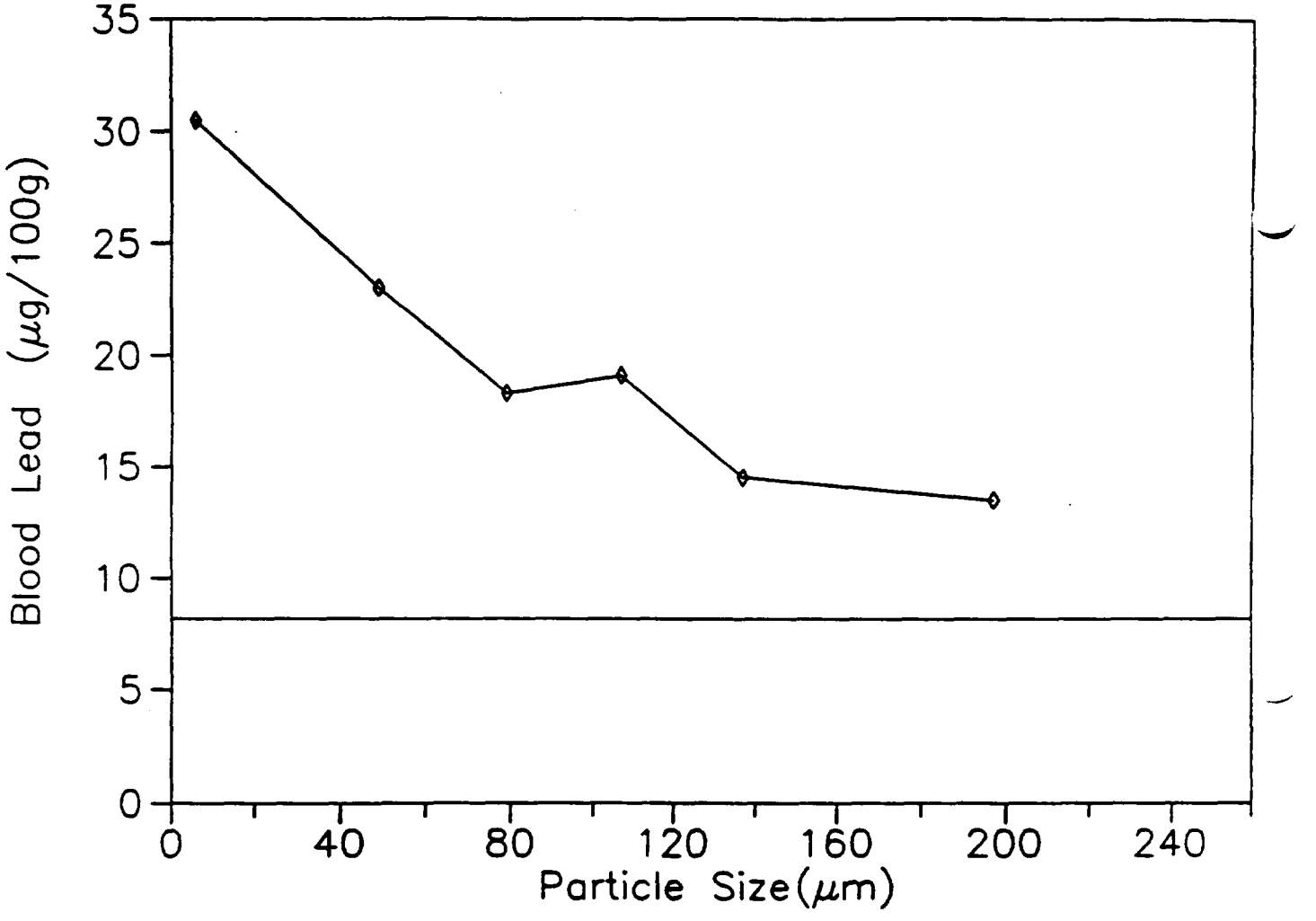



$r^2 = 0.18$, significant at $p = 0.05$
Slope* = 0.34 μg lead/dl blood per μg lead/$m^3$ air
* Corrected for an 8 hour/day, 5 day/week to a 24 hour continuous exposure.

Reference: Roy, 1977

Figure 5

**Effect of Particle Size on Lead Absorption from the Rat Gut**

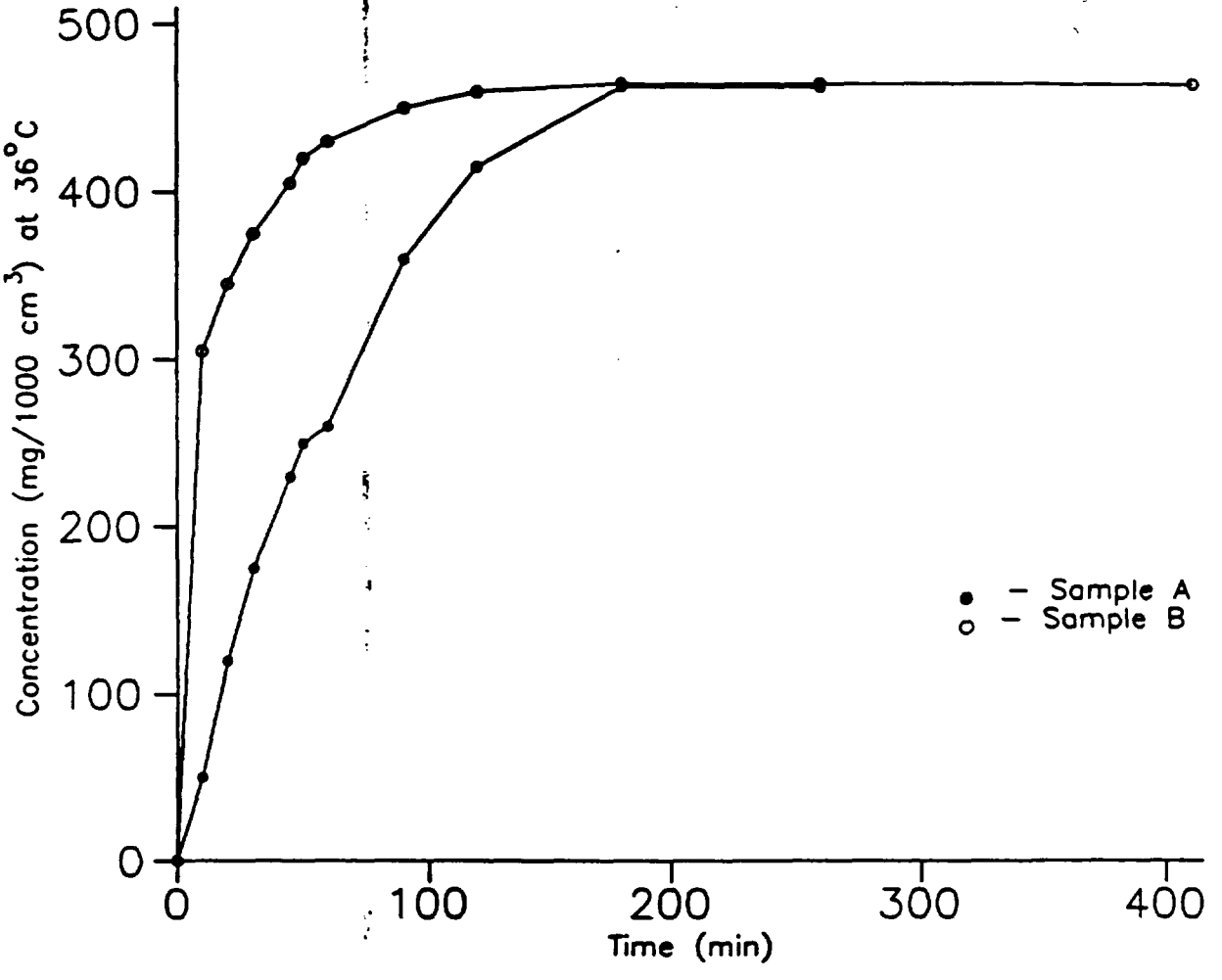



Experiment: Rats, metallic lead particles added to food for 48 hours.

Reference: Barltrop and Meek, 1979

Figure 6

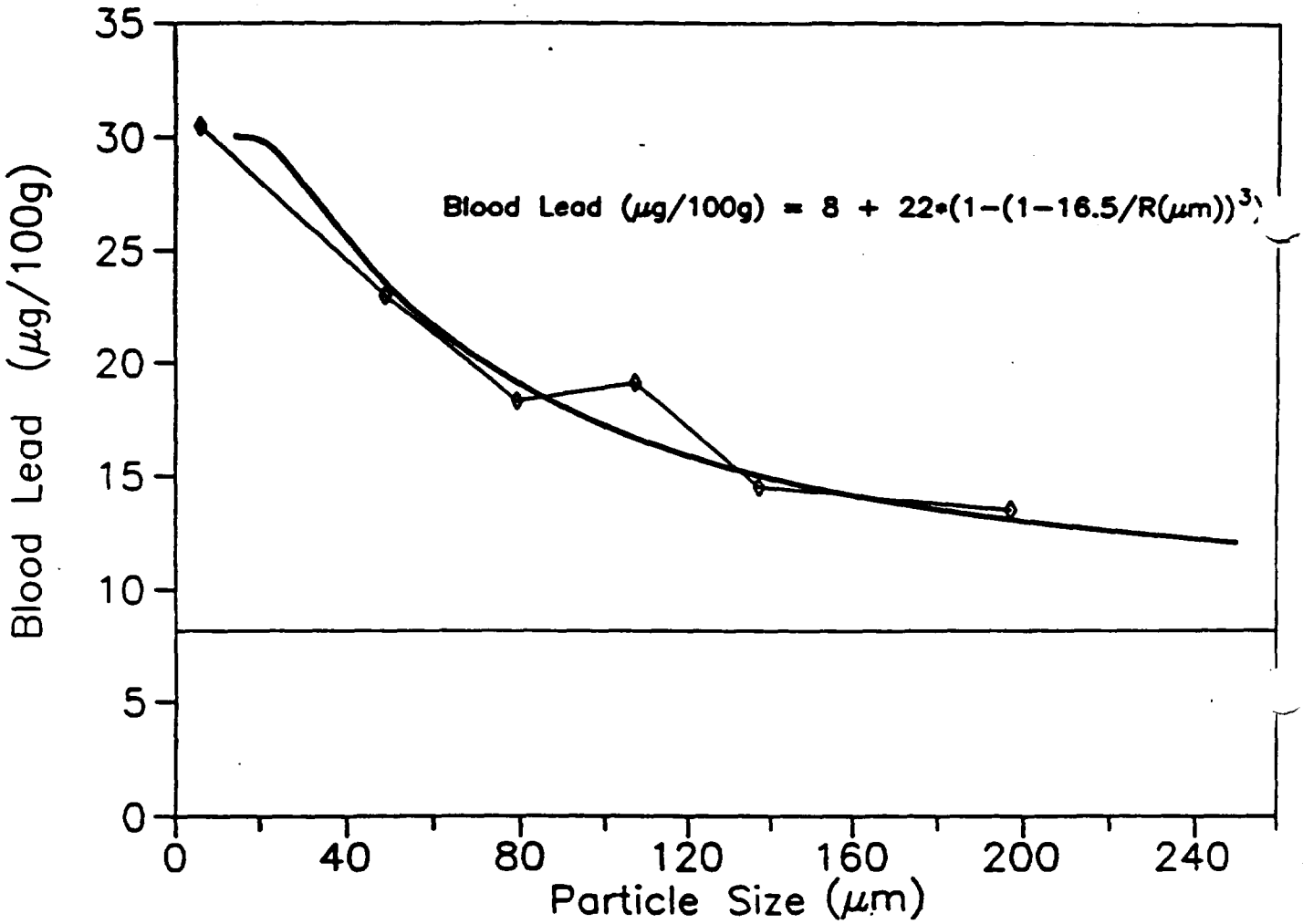
Variation with particle size of the rate of dissolution of lead sulphide in gastric fluid.
Sample A, 100 +/− 20 $\mu$; Sample B, 30 +/− 20 $\mu$




Reference: Healy et al., 1982

Figure 7

**Effect of Particle Size on Lead Absorption from the Rat Gut**




**Experiment:** Rats, metallic lead particles added to food for 48 hours.

**Reference:** Barltrop and Meek, 1979